



BLOOD-PRESSURE

FROM THE CLINICAL STANDPOINT

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PREFACE TO SECOND EDITION

IN the preparation of the second edition of this little work the author has adhered to the plan originally outlined by avoiding wherever possible unnecessary discussion and the introduction of theoretic and inconclusive data.

More careful methods of clinical study have enabled the author to introduce a larger amount of clinical material including many new charts, illustrative of various types of blood-pressure changes, from which it is hoped that the reader may be able to acquire a more practical view of the subject.

In the following pages the purely scientific investigator should not be too critical of the conclusions drawn but should allow the author considerable latitude, as, throughout, the chief effort has been to reduce a very large and complicated subject to a practical working basis, one which may be applied to everyday conditions, which after all is the chief concern of the practitioner.

For valuable assistance rendered in the preparation of this edition the author desires particularly to thank Drs. George P. Müller, Francis J. Dever and Brooke M. Anspach.

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PREFACE

THE past few years have marked a rapid rise in the clinical value of the sphygmomanometer. This instrument is now a part of the armamentarium of almost every physician. It is opportune therefore that a book of moderate size should be produced containing in concise form, a résumé of the clinical and experimental work which has led to present popularity of the blood-pressure test.

In the following pages, the author has endeavored to present in easily accessible form, the pith of medical literature bearing on blood-pressure studies in their relation to medicine, not only in cardio-vascular and renal conditions, but also in many diseases in which clinical observation has shown the information obtained by the sphygmomanometer to be of value.

It has been thought advisable to devote a number of pages to the discussion of the circulation and its relation to the blood-pressure, together with the various methods employed in sphygmomanometry, to acquaint the practitioner with the theory of this procedure, so that deductions from his observations may be of greatest value.

The writer is indebted to a large number of authors for much of the material contained in this work, which has been obtained largely from the medical literature of the last seven or eight years. Whenever practical the full reference is given in the text, so that if desired, the facts contained in this little book may be supplemented by a study of the original.

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BLOOD-PRESSURE

INTRODUCTION

The study of blood-pressure is unique in that it is the only subject employing a method of precision which enjoys an almost universal field of application, and that, unlike other clinical investigations which require special apparatus, its employment is not confined to the internist and the laboratory worker. The blood-pressure test being easily applied and the results more than satisfactory, it has rightly found general favor at the hands of widely varying branches of the healing art.

This great and sudden popularity has had certain drawbacks, as a tendency has been created in some directions to jump at extreme conclusions regarding the value or non-value of the test, so that either unlimited reliance has been placed in the findings to the elimination of other perhaps equally important data, or the value of the study of blood-pressure has not been adequately considered by other clinicians who as a result have failed to give their patients the benefit of its undoubted advantages.

In spite of the comparatively recent development of this test in its present form, there has already accumulated a most extensive literature making it almost impossible to keep abreast with it. The author has in this work endeavored to collate and sift out the many points of value on blood-pressure found in recent and current literature,

and to place them in such shape that the busy practitioner may speedily acquire a general view of the subject. In so doing, the author has been forced, for reasons of economy of space, to limit the discussion to certain fixed lines. Thus, no attempt has been made to devote great space to a consideration of the theoretical and experimental sides of the subject, either from the physiologic or the pathologic standpoint, except in so far as they have an immediate and practical application. It was thought advisable, however, to insert a number of extra references for the benefit of those who wish to consider the subject more comprehensively.

Neither has unnecessary space been occupied in describing the large number of blood-pressure testing instruments, many of which have become either obsolete or nearly so, while others vary so little in character, design, or in operating principle that detailed description was deemed unnecessary. Others again are so complicated in mechanism or mode of operation as to render them practically without value in clinical medicine.

The preparation of this revised work has entailed the reading and sifting of an immense amount of "literature" for which due credit has been given to the authors, whenever practical; while the great volume of material to review has increased the probability of inadvertent omissions, which are, it is believed, neither many or serious.

At the same time rules for the admission of material have had at times to be closely drawn in the interests of brevity, that perhaps some omissions have been made which may perhaps be deplored by the reader.

CHAPTER I

PHYSIOLOGY

THE CIRCULATION

As practice makes perfect, so has the extended and universal application of the sphygmomanometer perfected and reduced to a practical basis a large amount of theoretical and conjectural material bearing on blood-pressure which, until recently, was without scientific foundation. For this reason, in order to gain a proper appreciation of the function of the sphygmomanometer and the relation of blood-pressure readings of systolic, diastolic and pulse pressure, to the changing conditions in the cardiovascular system which this study reveals, it is primarily essential to possess a working knowledge of the theory of hydrostatics, and its relation to the human physiology, together with its modification by physical and vital conditions surrounding the human circulation both in health and disease. It is only by the knowledge of at least the essentials of this subject, that a proper appreciation of the factors controlling the cardiovascular system in both health and disease and the relation of this system to the kidneys and other organs closely associated with it can be had. It is proper, therefore, to preface a clinical study of blood-pressure with a brief general discussion of the circulation, even though to so do may seem to some to be a needless waste of time and space.

General Considerations.—The maintenance of a normal circulation is essential to health. Permanent (and transient also) alterations in the circulation are either the result of disease, or result in it. The action of the heart is a vital function like respiration, for if the heart should cease to beat even for a very short space of time, the circulation would fail, and the individual would die. The heart must not only act continuously, but it must also perform its function in an approximately normal manner in order to maintain a normal circulation which is essential to health.

Conditions affecting the action of the heart are frequently shown by alterations in the circulation, and variations in the circulation may often be shown by changes in the heart's action. Failure of the circulation is accompanied by a gradual diminution in blood-pressure, until it becomes insufficient to maintain body nutrition and offers insufficient resistance for a normal action of the heart. The two conditions, heart action and a maintained circulatory equilibrium, are in every way interdependent. They can neither be separated, nor considered intelligently, one apart from the other. This serves to emphasize the importance of a study of the circulation, not only in an investigation of circulatory diseases and cardiac affections but also in the study of diseases of practically any part of the body including those of infectious and metabolic origin.

We are for the most part indebted to Harvey¹ for discovering and demonstrating the true function of the

¹ See Camac's "Epoch-making Contributions to Medicine and Surgery," Philadelphia, 1909.

heart as the mainspring of the circulation. In 1616, Wm. Harvey stated that "a perpetual movement of the blood in a circle is caused by the beat of the heart." It is evident that Harvey's conception was the true one, as it forms the basis of our modern theory of circulatory physiology and pathology. It is of interest to note, however, that investigations of ancient manuscripts, bearing on medicine, show that some knowledge of the circulation of the blood was possessed by the ancient Egyptians as shown by references to the heart, and the use of the word "circulate" in the Ebers papyrus. In this connection an interesting review "The Advance in Knowledge Regarding the Circulation of the Blood" has recently been published by Dr. Geo. Wm. Norris.

The Normal Circulation.—Broadly speaking, it may be said that the term normal circulation presupposes a normal blood-pressure maintained by a normally acting heart with normal vessels and, as a corollary, abnormal blood-pressure is an indication of some abnormality in the circulation. Were this in every sense true in practice, the whole study of blood-pressure and its relation to the pathology of the circulation would be a simple and open book. When viewed from the standpoint of the human organism, involving as it does many complex and inter-related mechanisms, which are in turn affected both by internal and external conditions it is evident that so many factors must enter into this consideration, that we are forced to the conclusion that the study is a complicated one, which can only be understood by a most careful consideration of the sum of many internal and external influences affecting the cardiovascular system and the kidneys.

COURSE OF THE CIRCULATION

In the human body we may trace the course of a given particle of blood as it leaves the right ventricle until, having traversed the entire cardiovascular system, it returns to the starting-point. Referring to Fig. 1, we find the

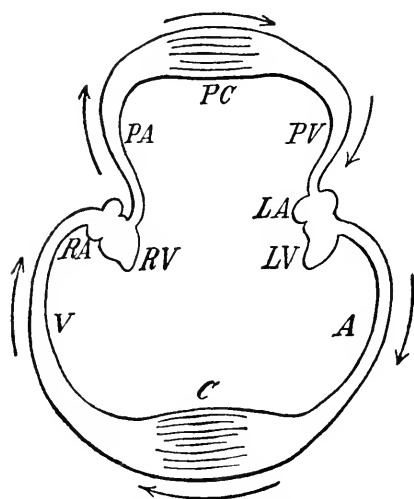


FIG. 1.—General diagram of the circulation: the arrows indicate the course of the blood: *PA*, pulmonary artery; *PC*, pulmonary capillaries; *PV*, pulmonary veins; *LA*, left auricle; *LV*, left ventricle; *A*, systemic arteries; *C*, systemic capillaries; *V*, systemic veins; *RA*, right auricle; *RV*, right ventricle. (John G. Curtis in "American Text-book of Physiology.")

course of the blood to be as follows: From the trunk of the pulmonary artery through a succession of arterial branches into the capillaries of the lungs, from there through the several branches of the pulmonary vein to the left auricle of the heart, thence through the mitral valve to the left ventricle, then by way of the aortic valve to the aorta and the general arterial tree until it finally reaches the capillaries. From the capillaries into the veins back toward the heart

through the venæ cavæ and into the right ventricle, through the pulmonary valve into the pulmonary artery where the tracing of the circuit began.

The physiologic factors controlling the normal changes in the circulation have been carefully studied and sum-

marized by Gibson¹ who states that the circulation, in the higher animals including man, is based upon four primary groups of principles: (1) Mechanical arrangement; (2) chemical changes; (3) glandular secretions; and (4) adjustment of nervous agencies; and that these four principles are capable of most complicated interrelation. A proper adjustment between them is maintained in health and is responsible for a normal circulation.

1. Mechanical Arrangements.—We find that these comprise: (a) the vascular system, which is a closed series of tubes (arteries and veins) of varying diameters; (b) the heart, which mechanically, is an intermittent force pump, interposed between the largest veins on one side and the largest arteries on the other. This tubular system is partially interrupted at two points by a series of very minute vessels (c) the capillaries of the lungs and of the general circulation.

The condition of the arterial walls and the width of the arteries exercise considerable influence upon the flow of blood. If all the arteries were fully dilated it would be absolutely impossible for the heart to maintain the circulation, because the relatively small amount of blood in the body could not begin to completely fill the vessels.

The force by which the blood is driven from the right to the left side of the heart, through the capillaries which are related to the respiratory surface of the lung, is nearly all derived from the contraction of the muscular wall of the right ventricle. The force by which the blood is driven from the left side of the heart through the general circulation, including all the other capillaries in the body, is

¹ *Brit. Med. Jour.*, July 27, Vol. 2, No. 2691.

nearly all derived from the contractions of the muscular wall of the left ventricle. The contraction of the two ventricles is simultaneous. The force generated by the heart in maintaining the circulation is, to a subordinate degree, supplemented by the aspirating action of the chest wall during the respiratory act, by the pumping action of the skeletal muscles and by the elasticity or tone of the arteries themselves.

The usual systolic arterial blood-pressure, which varies between 110 and 140 mm. Hg., is much more than is actually necessary to drive blood from the arteries into the veins. This extra pressure has a function, however, which is seen whenever the arterioles of any organ or small area relax. If the arterial pressure was barely adequate to sustain a flow, a lowered resistance in any part would seriously drain other regions. The high head of pressure, therefore, serves to keep all parts properly supplied with blood, even if an especially active part of the body is making an unusual demand.

In order to better understand the mechanical arrangement of the cardiovascular system it is necessary first, to consider the science of hydrostatics as to its effect upon the circulatory system.

— Fluids are incompressible and the heart is an intermittent pump; therefore if the arteries were rigid and unyielding tubes, each increment of blood coming from the heart would be required to move all the blood in the whole arterial system, while during the heart-rest, all flow would cease. This would result in the intermittent development of pressure, accompanied by periods when it must fall to zero. Such a condition would be inimical to health,

as the proper nutrition and tension of the organs and tissues of the body would not be maintained. The arterial walls are, however, as already stated, not rigid but elastic and distensible, and are capable, therefore, of expanding under pressure to accommodate more fluid, while during diastole their elasticity and contractility tend to maintain pressure. This property gradually reduces the sharp intermittency of the flow in the arterial system, so that, as we pass outward from the heart, this feature becomes less marked and finally disappears before the capillaries are reached. Another factor enters here. This is the gradual tapering and extensive ramification of the arterial system. The length of vessels combined with their elasticity further aid in reducing this intermittency to a uniform current.

A third factor is the relatively large number and minutely small diameter of the capillaries. If the vessels were short and the tubes of large diameter the flow would pass into the veins intermittently. This is seen in certain pathologic conditions where we have a capillary pulse and a transmitted venous pulse.

In considering the capillaries, we find that they also assist in maintaining blood-pressure, for if the heart as a pump was large enough and the arteries short enough and the outlet large enough there would be no blood-pressure.

Starting with the arterial system as a closed system of tubes, including the heart, we find that as the heart begins to beat, blood is pumped into the arteries, and in its passage toward the capillaries it meets with resistance. This causes the pressure to rise in the arterial system. This increase in pressure brings into action the elasticity of the arterial walls, so that as the pressure rises the

arteries expand to accommodate an additional amount of blood, at the same time the blood-pressure rises; this increases the pressure in the capillary system and drives more blood into the veins in a given time. Blood-pressure will reach normal and be maintained there, when the amount of blood passing through the capillaries during a heart cycle equals the amount entering the aorta during systole. At this time, the power of the heart is exactly balanced by the resistance in the arterial system and a level of pressure is established.

The mechanical arrangement of the circulation, therefore, provides for the general distribution of blood to all parts of the body, through the agency of the heart, the arteries, the capillaries and the veins and, in addition, provides for the conversion of an intermittent cardiac output into a uniform flow in the confines of the arterial tree.

2. The Chemical Changes.—Our knowledge of the chemical changes taking place in the circulation and their relation to the control of blood-pressure is but limited. It involves a knowledge and consideration of the effect of the catalytic action on a number of substances of a complex organic nature, whose function it is, at least in part, to preserve the normal composition of the blood and to control and regulate the normal interchange of gases in the capillaries of the lungs, and of pabulum and waste materials in the systemic capillaries and tissue spaces.

The chemical regulation is probably maintained through the agency of certain substances, acting upon the vasomotor system and on the arterial lining. Thus the work of H. A. Stewart and S. C. Harvey appears to demonstrate

the presence of a vasodilator substance, of probably proteid nature, which is specific for the renal vessels. They state further that these substances appear to act both directly upon the muscular coat of the vessels, and also through the vasomotor centers of the medulla.

It has been suggested that the carbon dioxide and lactic acid liberated during muscular activity play a part in this regulating mechanism.

R. M. Pearce¹ experimenting with the extracts of kidneys of various animals has demonstrated a mixed chemical action, in that these extracts may either diminish or increase blood-pressure, thereby differing in their effect from extracts of the adrenals (see below). He also found that there was no difference in the effect of extracts of normal kidneys as compared with those which were the seat of different forms of nephritis. R. Mohr² has demonstrated that an increase in these substances may produce sudden and serious drops in pressure, as noted previously by Sabatowski.

Closely related to, and possibly dependent partly upon, chemical changes in the blood, is the effect of the interchange of fluids between the blood-vascular and the lymph-vascular systems and the tissues of the body. While this is undoubtedly in part controlled by the special vasomotor system, nevertheless it is not unreasonable to suppose that changes in the chemical composition of the blood affect the relative amount of and distribution of blood and lymph in the human body, while it is well known that abnormal substances, particularly bacterial

¹*Jour. Exp. Med.*, May, 1909.

²*Wien. klin. Wochen.*, May 16, 1912, xxv, 20.

toxins, and those of intestinal origin, have a most profound effect upon the blood-pressure level (see page 255).

3. Glandular Secretions.—The chromaffin system has of late offered a fruitful field for experimental research in many directions, among which the relations of the glands of internal secretion to both normal and abnormal blood-pressure have been exhaustively studied.¹ In spite of this experimental activity much remains to be done and many points need additional light before the complete relationship of the several glands comprising this system, to changes in the circulation, will be satisfactorily explained.

The adrenals, the pituitary, and the thyroid have been shown to occupy the most important rôles. Early experimental work in this direction, notably that of Hoskins, McClure, and Janeway and Park was unconclusive, chiefly because so little was then known of these organs and their chemico-vital effects, that the premises upon which the investigations were based were erroneous.

The clinical effects of the commercially prepared extracts of the thyroid, the pituitary and the adrenals on the circulation are well known (see pages 246–428) yet we are by no means assured that the action of these glands under normal conditions of secretion is the same either in character or degree.

The pituitary has been shown experimentally² to have

¹ Fawcett, Rodgers, Rahe and Beebe (*Am. Jour. Physiol.*, January, 1915, xxxvi, No. 2); Blackford and Landford (*N. Y. Med. Jour.*, Sept. 13, 1913); Jordan and Eyster (*Am. Jour. Physiol.*, 1911, xxix, p. 115); Lewis, Miller and Matthews (*Arch. Int. Med.*, June, 1911, p. 785); Glay and Cleret (*Jour. de Physiol. et de Path. Générale*, 1911, xiii, p. 928).

² Behrenroth, *Deut. Arch. f. klin. Med.*, cxiii, Nos. 3 and 4.

a decidedly mixed action, since the vasodilator or vasoconstrictor activity of the extract depends upon the portion of the gland employed.

As to the effect of the normal adrenal secretion, Hoskins and McPeck¹ have failed to show that massage of the normal adrenals, in narcotized dogs caused a rise in blood-pressure at all comparable to that following the ordinary employment of adrenalin in the usual doses. And they conclude that the adrenal secretion is not an immediate factor in normal blood-pressure maintenance.

On the other hand in high blood-pressure (from abnormal adrenal conditions) C. Quadroni² treated a number of cases of permanent high blood-pressure by exposing the adrenals to the X-ray and obtained a uniform reduction of the systolic pressure, thus confirming the experience of Zimmern.

It is also shown that the administration of thyroid does not invariably cause a reduction in blood-pressure even in cases where there is a marked elevation.

The recent work of Cannon and Nice³ demonstrated an increased adrenal output and consequent increase in blood-pressure in cats as result of excitement.

In this connection H. E. Waller⁴ advances the plausible theory of the interrelation of the thyroid and adrenal secretions, holding that he has proven by experimental work that such an interaction really exists and the normal

¹ R. G. Hoskins and C. McPeck, *Jour. A. M. A.*, June 7, 1913, Vol. LX, No. 23.

² *Riforma Medica*, Feb. 8, 1913, xxix, No. 6.

³ *Amer. Jour. Physiol.*, 1913, xxxii, 44.

⁴ *Practitioner*, London, May, 1913.

level of blood-pressure is maintained through the channels of secretion by varying amounts of the active substances of these two glands. He explains the blood-pressure variation in myxedema and early Graves disease upon this basis. That these substances are alone in control of the field is very doubtful, as recently Voegtlin and Macht¹ claim to have discovered a new vasoconstrictor substance from the blood of the adrenal cortex, whose physiologic action is somewhat similar to that of digitalis, while chemically it is akin to lipid cholesterin.

While these several theories may be in part reconciled to our present knowledge of the relations of blood-pressure in health and disease, they must not be accepted as conclusively proven. For we have no proof that the substances extracted from these glands ever normally enter the blood-stream in a chemical form that we are able to isolate them.

4. The Vasomotor Nervous Mechanism Controlling Blood-pressure.—*The Maintenance of Tonus.*—The condition of tonus may be defined as, a state of semicontraction of the several coats of the arterial walls, which is maintained through the activity of a complicated vasomotor mechanism. This condition of tonus is most important, since changes in the force and frequency in the heart's action, and variations in the total amount of blood in the body, or in the supply to any organ or part, would be at the mercy of every other part, unless controlled by the automatic contraction and dilatation of the vessels in other parts of the body. Such a mechanism is essential to the proper distribution of blood to all regions at all

¹Carl Voegtlin and David I. Macht, *Jour. A. M. A.*, December 13, 1913.

times. This condition in any individual part is known as *local vascular tone*, and is under the control of a system of nerve ganglia with subservient fibers, which are found in the middle coat of all arteries. The energy expended by these ganglia is manifested by a constant moderate contraction of the circular muscular coat of the artery—a contraction which is constantly opposed by the dilating force (lateral blood-pressure) within the vessel. An exact equipoise between these two forces never occurs, since each factor varies constantly; but in a state of health, neither one ever becomes permanently excessive. Considerable variations, however, in the local vascular tone are frequently observed. Thus, each organ is influenced to a certain degree by every other, since an increase in blood in one part must necessarily involve a decrease in other parts, the total amount of blood in the vessels remaining constant. Those of the splanchnic area are chiefly concerned in maintaining this balance, since, if local vasomotor control of these regions were, for any reason, abolished, the arteries and arterioles would dilate sufficiently to contain practically all the blood in the body. The result would be a condition similar to that following a *massive* hemorrhage.

Alterations in the heart's action are felt more quickly in some organs than in others and, thus the general blood-pressure for cardiac reasons may be varied sufficiently to cause secondary disturbances in local vascular tone.¹

The nervous mechanism which controls local vascular tone is a complex one, consisting not only of a set of local ganglia in relation to the vessels and connected with the

¹ C. J. Wiggers, *Jour. Exp. Med.*, January, 1914, xix, p. 1.

larger sympathetic ganglia; but also of centers in the spinal cord, which are in turn connected with higher centers in the brain. The brain centers in turn are complex, consisting of an automatic mechanism in the medulla, regulating the action of all the subordinate parts below it, and of a series of cortical centers whose function it is to stimulate or inhibit the medullary mechanism.¹

Vasoconstrictors.—These facts seem to warrant the conclusion that the energy expended by the local ganglia in holding the vessels in a state of constant moderate contraction, is derived from the central nervous system, primarily from the automatic center in the medulla, which in turn is reinforced by each of the secondary centers in the spinal cord and sympathetic ganglia; and also that, while the medullary centers control the entire body, the cord and sympathetic centers control only these parts with which they are especially related. Any injury or interruption in the impulse to one or more of these parts will produce a vascular dilation through interference with the transmission of vasoconstrictor impulses from within outward; and irritation of one or more of these parts may cause a contraction of the vessels by increasing the normal stimulus sent out to local ganglia by the vasoconstrictors. An example of the general action of such irritation is shown by the increasing pressure in cerebral hemorrhage or intracranial pressure variation due to abscess or tumor, and locally, in the vascular condition met in true migraine (Starr).

¹ M. Allan Starr, *Jour. A. M. A.*, Vol. liii, No. 3, July 17, 1909, from whose article much of the material relating to vasomotor influence has been taken.

Vasodilators.—The action thus far considered has been wholly of the vasoconstrictor kind, and the dilatation mentioned has been due to a cessation or reduction of the constrictor energy normally passing outward. This may be termed a *passive dilatation*. It is the kind produced by division of any one of the sympathetic ganglia.

Experiments have shown, however, that another kind of dilatation can be produced, traceable not to a mere cessation of constrictor impulses, but to an impulse of a positive nature sent out to local ganglia and resulting in a sudden suspension of their activity. Such an impulse is really inhibitory, arresting the action of the ganglia in spite of the continued stimuli sent to them from the central nervous system. The result is a dilatation of the arteries, produced by the blood-pressure within; this may be termed an *active dilatation*. An important difference has been established between these two sets of impulses, namely, that while the action of the former is constant, that of the latter is intermittent. Therefore they cannot be regarded as opponents of each other.

From the anatomic standpoint the vasodilators can be traced upward in a manner similar to those controlling the vasoconstrictors, and experimental research has demonstrated the exact course of these fibers in relation to the various parts of the central nervous system, it has also been demonstrated that they exist as separate nerves, sometimes running together, although they usually enter the spinal cord at different levels.

Vasomotor Reflexes.—Vasomotor reflexes are found in all parts of the body, examples of which are seen in the effect of a change of temperature, or of pain, upon local or general

blood-pressure, or in the color of the face and the size of the pupil. It is probable that the major vasomotor reflexes are of a chemical nature, the chemical substance often acting directly upon the lining of blood-vessels so that this subject is closely related to the two preceding, namely, the chemical and glandular activities (see page 26).

Having reviewed our present knowledge of the physiologic relations of blood-pressure and vasomotor tone, we can now approach the practical clinical side of this subject more intelligently as, through this knowledge we are more readily able to comprehend the complex changes occurring in blood-pressure in disease.

For practical purposes it will be found convenient to consider the maintenance of normal blood-pressure and the production of abnormal pressures, more from the clinical standpoint, while admitting them to be dependent upon these primary physiologic divisions. This is at least the most convenient because more capable of clinical demonstration and study.

From a clinical standpoint the factors concerned in the maintenance of circulatory equilibrium are:

First.—The energy of the heart.

Second.—Peripheral resistance.

Third.—Arterial tonus.

Fourth.—Volume of the blood.

Fifth.—Viscosity.

They appear to be related to the physiologic factors in the following manner:

The energy of the heart, to the mechanical arrangement, and to the nervous mechanism; peripheral resistance and arterial tonus to the chemical changes, glandular secre-

tions and the nervous mechanism. The volume of the blood to the chemical changes and nervous mechanism and the viscosity of the chemical composition.

One should not be led, in the consideration of this subject into the belief that these several factors are entirely separable. As a matter of fact they are all so interrelated that it is utterly impossible to decide where the effect of one ceases and that of another begins; since the normal circulation is made up of the sum of activities and of these factors, which all may and which do vary constantly, under both normal, and in pathologic conditions. While it might be theoretically possible for one factor alone to produce discernible alterations in the circulation, it is improbable that it ever does, as all are so closely related one to the other any definite alteration in one of them must result in change in others. We are, therefore, forced to a consideration of the subject as a whole, while acknowledging and endeavoring to isolate the predominating effect of one or more, in relation to the pathologic changes found in any given case.

CHAPTER II

TERMS AND DEFINITIONS EMPLOYED IN THE STUDY OF ARTERIAL PRESSURE

Before undertaking a study of the clinical relations of blood-pressure, with its many and complex variations of both physiologic and pathologic origin, it is essential to have a knowledge of the different terms now in common use in the study of blood-pressure, to be familiar with the several accepted methods of study and to have a practical conception of the relations of the several events in the cardiac cycle to the various factors concerned in the maintenance of normal arterial pressure. A brief review of these matters may be conveniently considered here.

BLOOD-PRESSURE

Beginning with a broad general significance, when the term blood-pressure was applied to almost any condition of arterial pressure in any part of the circulatory system. The word has gradually become restricted, so that at the present writing, unless qualified by defining adjectives, the term has become so indefinite, that it should not be employed. The term, to have any clinical significance, must be qualified by the proper adjective, which will indicate what particular part of this general subject is under consideration, thus: systolic blood-pressure; diastolic blood-pressure; pulse pressure; mean pressure; average pressure;

venous pressure; capillary pressure; intraventricular pressure, etc.

Definition.—When unqualified, the term blood-pressure is taken to mean the systolic blood-pressure as measured in the brachial artery, though it is advisable to be more specific in all discussions and writings upon the subject of pressures, and to apply carefully the proper qualifying words in order to afford more definite scientific and therefore more accurate information.

In the chapter on physiology it has been said that arterial pressure is subject to many and various modifying factors and that we know now that the arterial pressure is in a state of constant rhythmic fluctuation due to the intermittency of the cardiac systoles, and also that the arterial pressure in normal individuals at all times is dependent upon the proper correlation of five distinct and separately determinable factors (see page 34).

Our present nomenclature of arterial pressure includes a number of terms some of which will be briefly defined and discussed.

First.—Arterial pressure (either end or lateral) which is divided into:

Second.—(a) Systolic or maximal pressure (measured by end pressure) and

Third.—(b) Diastolic or minimal pressure (measured as lateral pressure) from which we determine.

Fourth.—The pulse pressure, range or amplitude.

Fifth.—Mean arterial pressure.

Sixth.—Average arterial pressure.

The Arterial Pressure.—This is found to differ according to the manner of its measurement; thus the pressure which

is exerted against the vessel wall while the current of blood is flowing unopposed by direct obstruction, is known as the lateral pressure; whereas the pressure exerted by the blood against any obstruction which reduces the lumen of the vessel is known as the end pressure. The latter exceeds the former when measured at any given point in the arterial tree and the difference between the two is the measure of the dynamic power of the heart which is involved in actually carrying on blood flow at this point.

In clinical sphygmomanometry, the methods employed yield the end systolic pressure and the lateral diastolic pressure, and it is these pressures that are to be inferred when encountered in blood-pressure discussion.

The Systolic or Maximal Pressure.—This term indicates the maximum force exerted by the systole of the heart. It is the transmitted intraventricular pressure. This pressure measured in the brachial artery while it is less than, nevertheless tends to approximate, the intracardiac pressure, and the lateral pressure in the aorta.¹ Thus when we say that the systolic pressure is equal to 125 mm. Hg., we mean that the end pressure in the artery at the point of obstruction by the sphygmomanometer cuff, is capable of sustaining a column of mercury of 125 mm. over and above the atmospheric pressure.

The Absolute Pressure.—To determine the absolute systolic pressure, it is necessary to add to the observed systolic pressure in mm. Hg., the actual barometric pressure as measured by the same scale, at the time of estimation. This point should not be forgotten, although such an

¹Louis M. Warfield, *Int. Med. Jour.*, xix, No. 10, October, 1912.

estimation is rarely employed except in physiologic and experimental investigations.

Accuracy of the Systolic Pressure Estimation.—The difference between the systolic pressure, determined by sphygmomanometry in the brachial artery, as compared with that found in the aorta by direct measurement in animals is so slight that, for practical purposes, it is ignored. Therefore, when the systolic pressure is ascertained in the brachial artery, it represents a satisfactory clinical measure of the pressure throughout the arterial system except in very small vessels.

The systolic blood-pressure may be divided into two distinct portions, which have a valuable clinical and pathologic significance of the measure of the force exerted by the heart. It is compounded of the pressure required to overcome the minimal pressure in the aorta (the measure of peripheral resistance) plus the force necessary to cause arterial flow and to sustain the circulation in normal equilibrium (heart work).

Diastolic or Minimal Arterial Pressure.—This term refers to the pressure found in the smaller arterials and transmitted backward to the general arterial tree. It is, therefore, the measure of peripheral resistance and represents the amount of pressure that the left ventricle must exert before it can reestablish the forward movement of blood in the arterial system.

As the blood goes farther and farther from the heart toward the smaller arterials situated on the confines of the arterial tree, the systolic and diastolic pressures tend to approximate each other, until when the capillaries are reached the rhythmic fluctuation is lost, and a uniform

flow of blood issues from the capillaries (see page 25). Thus being dependent upon a fairly constant factor (peripheral resistance), the diastolic pressure varies very little in the larger arteries, where it is a comparatively fixed factor as compared with the systolic pressure.

All other factors remaining unchanged, a high peripheral resistance means a high diastolic pressure and a low peripheral resistance means a low diastolic pressure. In other words, vasoconstriction raises and vasodilatation lowers diastolic pressure.

The Pulse Pressure, Range or Amplitude.—These synonymous terms are employed to designate the blood-pressure variation in the arterial tree, occurring at any given point during a cardiac cycle. It represents the actual head of pressure driving the blood toward the periphery and is the measure of the force exerted by the ventricle

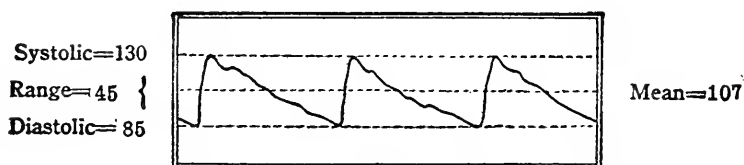


FIG. 2.—Normal pulse tracing: showing relation of systolic, diastolic, pulse pressure and mean. Pulse pressure equals 45.

over and above that required to overcome peripheral resistance (diastolic pressure). It becomes evident, therefore, that the pulse pressure may at any time be determined by subtracting the diastolic pressure from the systolic pressure and that without these observations the pulse pressure cannot be ascertained (see Fig. 2).

The determination of the pulse pressure is of great importance in clinical medicine, particularly in the prognosis

of many cardiovascular diseases, as it in all probability indicates alterations in heart-muscle efficiency not only in cardiac disease itself, but also in acute and chronic infections, cachectic states, etc. It is also used clinically to determine the actual velocity of blood flow.

The Mean Pressure.—There seems to be a degree of confusion and uncertainty in the minds of physicians and students regarding the terms *mean* and *average* as applied to blood-pressure findings. These terms are frequently employed synonymously, although as far as I have been able to ascertain they should not be used interchangeably even when coincidentally they may be found to be the same. I would emphasize this because, in a number of recent articles and in at least one recent text-book these terms have been loosely employed. Thus “by *mean pressure* is understood the *average pressure* (italics mine) at a given point; the mean pressure is not, however, the arithmetical mean between the systolic and diastolic pressure, because the pressure may rise for only a moment to the systolic level, falling very gradually to near the diastolic level and remaining near it throughout the greater part of the average cycle.”¹

Whatever the accepted usage of terms, average and mean may come to be, it is plain that they are used so ambiguously that they interfere with a proper understanding of a definite nomenclature.

Strictly speaking, we may obtain an average only by adding the sum of any number of similar units and dividing this sum by the number of units in the series, while we can obtain a mean only as between two similar units. We

¹ Norris, 1914 edition.

cannot therefore, determine the mean between the systolic and the diastolic pressure unless it be of two isolated observations, any more than plums could represent the

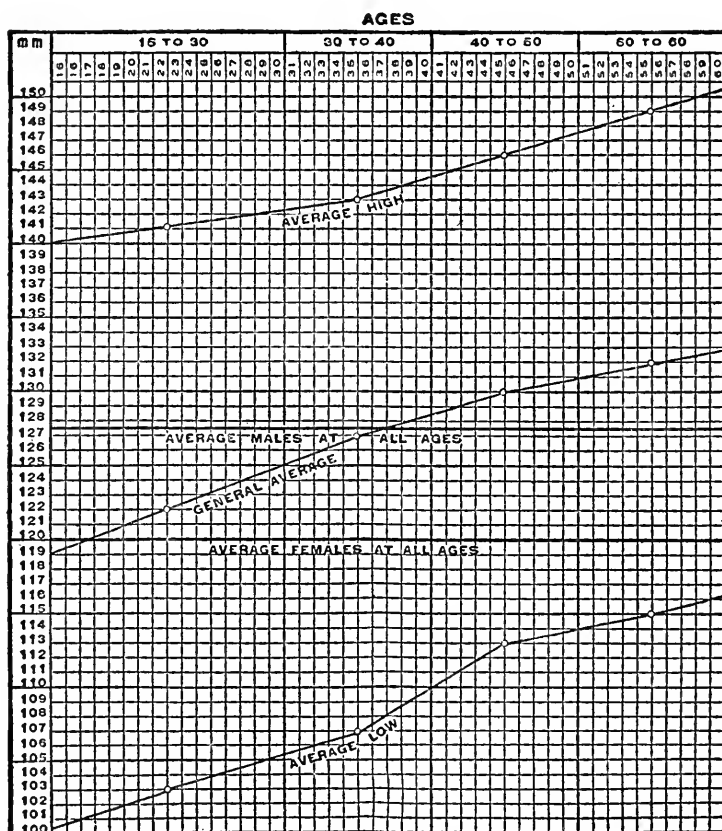


FIG. 3.—Woley's chart showing effect of age on blood-pressure, giving mean, high and low average.

mean between peaches and pears (see average pressure figures in Woley's table, Fig. 3). The only possible way of obtaining the mean pressure, either systolic or diastolic, in a series of observations, as for example to determine

the mean systolic and the mean diastolic pressure for a given age, would be to pair all the observations of the systolic pressure, making each pair the extremes, after having eliminated at each calculation the most widely separated pair. The result of the means obtained in this way would be a series of figures approximating each other, but not necessarily identical. This series of figures could then be added and an *average mean* obtained, which would in no way except by accident resemble the *average*. The same process carried out upon the diastolic pressure observations would produce an *average mean* diastolic pressure and from these a general mean pressure might, if advisable, be determined, and such a figure would not, except accidentally, be similar to half the pulse pressure added to the diastolic of any observation in the series.

The Average Pressure.—In sphygmomanometry, we depend on the average pressure either systolic, diastolic or pulse pressure as the most practical way of estimating the limits of normal variation. This will be referred to in the chapter upon normal pressures and their variation (see page 112).

The value and clinical significance of the mean pressure is yet to be determined.

THE CAUSES OF ARTERIAL PRESSURE AND THE FACTORS INVOLVED IN ITS MAINTENANCE

We believe the arterial pressure to be dependent upon five separate, distinct and individually variable factors.

These several factors are found to vary continually under normal conditions while in pathologic states they may and do undergo great and permanent changes, which are

reflected, as either transient or permanent variations in blood-pressure.

Arterial blood-pressure at any given time is the sum of these factors plus their reciprocal relations.

Not only may one vary independently of the other, but they are capable also of most complicated interaction.

1. The Heart Energy.—The heart is a force pump of intermittent action and is the most important factor in the circulation as it is the fountain head of all energy. But of itself it is powerless to maintain the circulation without the supplementary and supporting factors above mentioned. Thus while the heart supplies the force which causes the flow of blood, it is the peripheral resistance and arterial elasticity which are chiefly concerned with the maintenance of an adequate blood-pressure during the diastolic pause, while the heart is resting, which aggregates according to Brunton¹ on the average in the normal circulation, thirteen hours out of the twenty-four.

The left ventricle during systole forces the column of the blood into the arterial system while during diastole the blood is distributed forward throughout the arterial tree.

Any increase in the rapidity in the discharge of blood from the heart (increase in volume output per minute) will, the other factors remaining constant, cause a rise in systolic blood-pressure. Conversely any diminution in the pulse rate or volume output will cause a reduction in systolic blood-pressure. On the other hand a compensatory relation between the heart rate and the volume output may permit either or both (if inversely) to be altered without any appreciable change in blood-pressure.

¹ Lauder Brunton, *Brit. Med. Jour.*, Nov. 5, 1910.

2. Peripheral Resistance.—Peripheral resistance is that factor, ever present in the circulatory system, which tends to retard and prevent the forward movement of the circulating blood. In the human body this is a complicated factor, being dependent in part upon (*a*) the diminishing diameters of the conducting tubes, particularly in the arterioles and capillaries, (*b*) internal friction, (*c*) the length of the vessels and (*d*) the innumerable branching of the arterial tree.

It is obvious that any obstruction, even if partial, at the outlet of any distensible tube will increase the lateral pressure exerted by the fluid in that tube. A familiar example of this is the effect of the adjustable nozzle used on the garden hose. The same physical law holds good for the arterial system, except that the intermittency of flow is a complicating factor, which includes the interrelation of two variables—the systolic and the diastolic pressures. The result of these factors is to produce a condition in which increased peripheral resistance (increased vasomotor tone) will increase systolic pressure while a diminished peripheral resistance will lower both systolic and diastolic pressure. This change invariably occurs unless some compensating factor modifies the force of the heart (energy index, page 140).

The other factors, including length of tube and internal friction, being practically constant may be safely omitted in clinical considerations, without introducing any serious element of error (for further discussion of peripheral resistance and vasomotor tone, see Chapter VII, page 138).

Elasticity and Contractility of the Vessel Wall.—The elasticity and contractility of the blood-vessel walls is

dependent upon the circular muscular coat and the elastic lamina. Were it not for this quality of the arteries, the heart would be called upon to do a great deal of unnecessary work which would absorb a vast amount of valuable energy, while the flow of blood throughout the arterial system would be intermittent, as the heart at each beat would be required, in order to complete its systole, to drive the whole volume of blood forward and through the capillaries—a condition obviously incompatible with normal physiology in the body.

Thus during each systole the heart muscle performs work in maintaining arterial flow against peripheral resistance. A large part of this work, which is sudden and explosive (at systole), is compensated for by the expansion of the arterial walls.

A part of the manifest energy of the heart thus becomes for a time potential in the stretched fibers of the arterial wall. The moment that a systole is at an end, the stretched fibers recoil and continue the work of the heart in maintaining the arterial flow against peripheral resistance.

As this potential energy becomes expended the systolic pressure gradually falls and would eventually reach zero were it not for peripheral resistance plus the rhythmically recurring cardiac systole which causes the pressure to rise again.

The laminal elasticity of the vessels is very perfect and is capable of standing a pressure greater than by any chance could possibly be developed during life. According to Janeway,¹ quoting Gréhant and Quinguard, the carotid artery of a dog is capable of withstanding a pressure,

¹ *Loc. cit.*, p. 24.

twenty times greater than the normal blood-pressure without tearing. For the human carotid the lowest pressure at which rupture occurs is 1.29 meters of mercury, at least eight times the ordinary carotid pressure of the normal circulating blood.

4. Total Volume of the Blood.—Compared with the capacity of the arteries, capillaries and veins combined, the total volume of blood is surprisingly small, being variously estimated to be from one-twelfth to one-fifteenth of the body weight. In the normal individual the cubic capacity of the vascular system is so reduced by vasomotor control (see page 30) that the blood at all times is maintained under a considerable pressure.

While a certain amount of blood, probably about three-fourths that of the normal volume, is necessary to support the circulation, still it has been found that a large amount of blood can be withdrawn without interrupting cardiac action (see Venesection, page 460) and further that the pressure rapidly returns to a point at or near normal soon after the cessation of the hemorrhage. On the other hand, Worm Müller has shown that an amount of fluid greater than the total blood volume of the body can be transfused into the vessels, without increasing the blood-pressure above a point frequently reached under normal conditions. Therefore, it would seem that, except for great changes, the volume of the circulating blood has only a slight and temporary influence on normal blood-pressure.

5. The Viscosity of the Blood.—This factor has until recently been almost entirely ignored in observations and discussions of the normal and pathologic variations in

blood-pressure. It is, however, evidently a most important factor in determining peripheral resistance. This has been brought out by Allbutt¹ who states "that it is evident that friction in the arterial tree must multiply with every increase in viscosity as it has been demonstrated that nearly 200 times more of the heart's energy is expended in overcoming friction than is required in maintaining the velocity of the blood-stream." Variation in this factor, even when slight, must affect enormously the resistance offered to the passage of blood through the arterial system and therefore must profoundly affect the blood-pressure, so that we should follow most carefully any variation however small, in the viscosity of the circulating fluid.

Determann has found in plethoric persons, with high blood-pressure an increase in viscosity, while A. Martinet has shown that in normal cardiovascular conditions the blood-pressure parallels the viscosity. It will probably be found, as experimentation is carried further, that the viscosity of the blood is a most important factor in affecting blood-pressure, and that the development of methods for its modification or control will mark an epoch in the study and treatment of diseases involving blood-pressure changes.

When the viscosity is normal or below, as in renal arteriosclerosis for example, the blood-pressure will be high. On the other hand, if the viscosity is high, while the blood-pressure is normal or below, the pulse will be weak. Martinet has endeavored to build up a clinical picture upon this basis, in which such symptoms as cold hands and

¹ Clifford Allbutt, *Quarterly Jour. Med.*, 1910, p. 242.

feet, congestion of the liver, varices and various cardiovascular disturbances predominate.

The Pulse.—From our knowledge of the action of the heart, we know that blood is forced into the aorta at regular intervals, and that each charge of blood entering the aorta is felt throughout the arterial system in the form of a wave which is styled the pulse and which may be felt as a rhythmically recurring impulse (due to transitory increase in size of the vessel) in all palpable arteries.

The propagation of this wave throughout the arterial system implies a change in diameter of the vessel with a resulting stretching of the vessel wall (see Elasticity, page 45) caused by the increased increment of blood entering it. This further stretching of an already stretched vessel wall can occur only through an increase in pressure within the vessel sufficient to cause the increased diameter of the vessel which is left under the finger. It is a self-evident fact, then that there occurs alternately, in regular rhythmic cycle, a rise and fall in blood-pressure throughout the arterial system. Corresponding to the ventricular systole and diastole, the highest and lowest point of this change in pressure are termed respectively, systolic blood-pressure and diastolic blood-pressure (See Fig. 2).

CHAPTER III

THE PRINCIPLE OF THE SPHYGMOMANOMETER

THE INSTRUMENTAL ESTIMATION OF BLOOD-PRESSURE

The basis of modern sphygmomanometry is founded upon circular pneumatic compression, without which the modern sphygmomanometer could not have been developed, and the immense value of the results of blood-pressure study might still be unknown to clinical medicine.

The direct method of the physiologist is not applicable, as it would require a direct communication between the blood-vessel and the tube leading to the manometer, and is therefore not practical for the consulting room even if tolerated by the patient.

Circular Compression.—It remained for Riva-Rocci¹ and Hill and Barnard² each working independently of the other, to substitute the arm-encircling cuff for the uncertain and inaccurate pelote of the early investigators.

By means of the encircling arm-band (either full or partial) the pressure produced within the hollow inflatable rubber portion is exerted equally from every direction against the artery. Physiologic experiment has shown this method to be accurate, as the tissues intervening between the arm-band and the artery offer a negligible

¹ *Gazz. Med. di Torino*, 1896, Nos. 50 and 51.

² Leonard Hill and H. Barnard, *Brit. Med. Jour.*, 1897, Vol. 2, p. 904.

resistance,¹ so that the readings obtained, indirectly through the tissues, by the modern method, agree very closely with those obtained by the direct method.

Character of Cuff.—It has already been said that the width of the tubular cuff influences to a significant degree the readings obtained. Janeway has found in high-pressure cases that there exists a difference of as much as 60 mm. between the 12-cm. and the 5-cm. arm-band.² This is

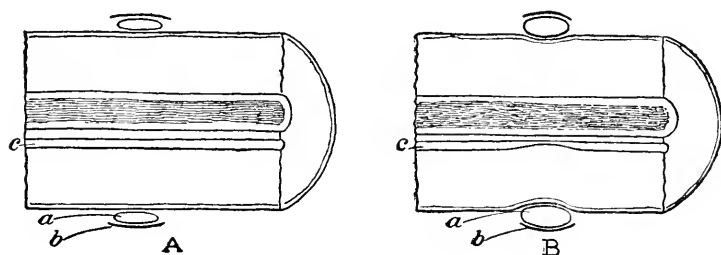


FIG. 4.—A. Schematic section of arm, showing narrow arm-band (*a*) with retaining device, (*b*) before inflation artery (*c*). B. Same, showing change in form of compression band (*a*), after inflation, artery (*c*) compressed. Note great change in form and increase in circumference of compression bag. This change occurred only at the expense of a measurable amount of pressure.

easily explained by noting the change which occurs within a narrow (2 in.) and a wide ($4\frac{1}{2}$ in.) cuff during inflation under a rigid retaining device—reference to the accompanying illustration will aid the explanation. Fig. 4 shows a narrow armlet which allows insufficient material to indent

¹ Von Recklinghausen, *Arch. f. Exp. Pathol. u. Pharmacol.*, 1901, xlvii, 78; Gumprecht, *Zeitsch. f. klin. Med.*, 1900, xxxv; and Hensen, *Deutsch. Arch. f. klin. Med.*, 1900, xlvii, p. 4.

² T. C. Janeway, "Clinical Study of Blood-pressure," 1907, D. Appleton & Co.

the tissues and compress the vessel without requiring additional pressure to expand the rubber bag, this amount being registered on the scale of the sphygmomanometer in addition to that required to compress the vessel. Chamberlain¹ has determined that this amount of error on an arm of average size is 8 mm. or more.

Fig. 5 shows wide arm-band (A) before compression and (B) after compression, where the change in form of the rubber portion is insufficient to exert pressure beside that required to indent the tissues and compress the vessel.

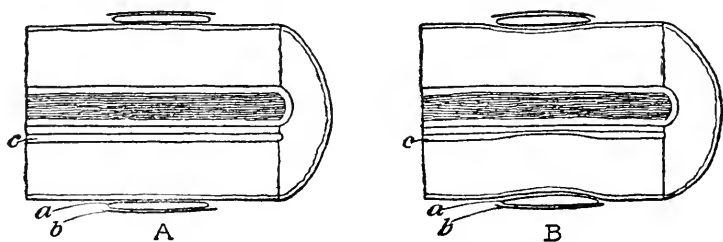


FIG. 5.—A. Schematic section of arm showing wide arm-band (a) with retaining device (b) and artery (c) before inflation. B. Same, showing change in form of compression bag (a) after inflation artery (c) compressed, note slight change in form of compression bag, insufficient to exert any additional force than that required to compress artery.

Influence of the Vessel Wall.—Upon this subject authorities differ. The early experiments of v. Basch² show that the resistance of a normal radial artery to closure scarcely amounts to 1 mm.

Russell³ takes the opposite stand when he says, "I cannot but think that those who have thought that the

¹ Chamberlain, *Philippine Jour. of Sci.*, vi, Sec. B, December, 1911.

² *Berlin. klin. Wochen.*, xxiv, 1887.

³ Wm. Russell, "Arterial Hypertension, Arteriosclerosis and Blood-pressure," J. B. Lippincott Co., 1908, p. 52.

arterial wall is negligible, have not had the data necessary to a correct opinion."

Hoover¹ recently attempted to prove Russell's contention by submerging an arm or leg in an ice-filled trough and demonstrating the changes in arterial pressure caused thereby. In this work Hoover apparently failed to recognize the well-known effect of peripheral constriction upon the proximal portion of the vessel (*i.e.*, central to the constriction) which, in his experiments is undoubtedly the cause of the rise in pressure; it therefore cannot be ascribed to change in the condition of the arterial wall.

Janeway² and later Janeway and Park³ believe the element of error to be greater than was formerly supposed, as they found that the resistance offered by the arterial wall in adults with normal pressures to be more than 1 or 2 mm. Hg. while they were able to demonstrate that this rarely equalled more than 10 mm. Hg. Practically this difference may be discounted as in adults the factor is a comparatively fixed one, and being ever present may be clinically ignored, and also because it is no more than the spontaneous variation occurring in any individual from time to time (often within a few minutes). In children the factor is entirely negligible.

Arteriosclerosis, according to Janeway and Park,⁴ even when marked, increases but moderately the normal resistance to compression. By actual experiment they found that it did not exceed 17 mm. Hg.

¹ C. F. Hoover, *Jour. A. M. A.*, Vol. lv. No. 10, Sept. 3, 1910.

² "Clinical Study of Blood-pressure," 1st edition, 1907, p. 61.

³ *Arch. Int. Med.*, No. 6, 1910.

⁴ *Loc. cit.*

From the data at hand, we may conclude that the vessel wall, as a factor, need not be considered from a clinical standpoint, as any resistance which could be offered by a vessel even markedly sclerosed, would be insignificant when compared to the alterations in pressure often occurring within the vessel in disease. I submit as further proof the many high-pressure cases met here but little change can be demonstrated in the superficial vessels, while on the other hand, cases occur whose superficial vessels, as far as they can be digitally traced, are absolutely rigid and yet blood-pressure by palpation may never register over 110 mm.

Influence of Other Intervening Structures.—Vital tissue is perfectly elastic; therefore any pressure applied to the surface of a living body will be transmitted directly to the underlying structure without loss of force.

It would seem, therefore, safe to assume that both the vessel wall and the intervening tissues, as definite factors which modify blood-pressure readings, can be absolutely eliminated, at least clinically, because all pressures are read through them and so, as they are always included in the estimation, they can be ignored.

THE ESTIMATION OF BLOOD-PRESSURE

No one will deny at this time the great importance of accurate blood-pressure estimation, and the signal value that clinical medicine has derived from the modern sphygmomanometer. Neither will anyone be deluded into believing that he or anyone else is able, by tactile sense alone, to determine the relative height of blood-pressure with anything like the same degree of accuracy as is attained

by the modern sphygmomanometer. While it may be true that extremes in pressure might be recognized, it is also true that the smaller variations, which are after all often the most important clinically, cannot be detected with any degree of certainty, if at all.

We will consider therefore, that no further argument is necessary, but will assume it to be an accepted fact that the accurate estimation of arterial tension is to be accomplished only by means of sphygmomanometry.

With the older instruments it was possible to roughly estimate the systolic pressure. At the present time approximate estimations are no longer sufficient as we now believe it essential to determine with accuracy not only the systolic pressure but also the diastolic and pulse pressures, as well. Experience has taught us that while the systolic pressure is important, it is nevertheless only a part, and frequently but a small part of the information sought.

It, therefore, becomes extremely important to consider clinically the accepted methods of sphygmomanometry and to possess a working knowledge of the methods and instruments employed.

There are a large number of instruments, mostly of very recent devising, which may be relied upon in competent hands for this estimation, and the choice between them is largely a personal matter. So that in the pages to follow all statements referring to specific instruments are intended only to supply a certain amount of working knowledge, obtained through actual experience, which may save the reader unnecessary experimentation when selecting an instrument.

METHOD OF EMPLOYING THE MODERN SPHYGMOMANOMETER

Having already considered the subject of blood-pressure in the abstract and having reviewed the criteria surrounding the proper performance of the study, we are now in a position to consider the actual clinical employment of the sphygmomanometer.

All modern clinical instruments are operated in conjunction with the elastic cuff or arm-band, which after encircling one of the extremities is inflated, so that by compression it will obliterate the arterial pulse. The measure of air pressure at the moment of complete obstruction is the measure of the systolic pressure. This has already been shown on page 50 to approximate the actual arterial pressure, as determined simultaneously by the direct introduction of a canula, so closely that the difference (a few mm. Hg) may be ignored. These readings if made with any modern type of instrument are comparable, as they all give the pressure readings in terms of millimeters of mercury. The several forms of apparatus will be discussed in detail later. Except for the style and construction of the manometer there is very little difference in the several apparatus either as regards their *modus operandi* or their clinical use, the only differences being slight variations in minor details such as the material forming arm-band, the location of tubes and other attachments, etc.

SUGGESTIONS TO BE FOLLOWED AND PRECAUTIONS TO BE OBSERVED WHILE USING THE SPHYGMOMANOMETER

Position of the Patient.—Whether the observation is made in the reclining or sitting posture will be determined

by the nature of the case and by exigencies of practice. In the critically ill the horizontal posture is preferable, although it will not always be found convenient or possible in the presence of orthopnea, while in ambulatory cases it will not always be found convenient to employ the horizontal. One point to be borne in mind is that for purposes of comparison it is essential, whenever possible, to make all subsequent observations in a case in the same posture as was the first. At all events the location of the arm-band, irrespective of the patient's posture, should be at the heart-level, thus eliminating the error due to gravity. Under all circumstances the patient should be in a comfortable position, and one favoring muscular relaxation.

Application of Cuff.—The cuff is usually applied to the arm, above the elbow and should be maintained at the heart-level, Fig. 6. It should be applied directly to the bared arm or over very thin coverings and wrapped firmly; this will avoid the unnecessary delay required to fully inflate a loosely applied arm-band. The arm-band should not exert pressure. This point is also of importance in using any method other than the auscultatory, since the greater the volume of confined air the less marked will be the rhythmic impulse transmitted to the manometer.

Time of Observation.—Whenever possible observations should be made at about the same time of day, and in the same relation to the taking of food. Observations should not be made during periods of excitement, or after exercise, or in periods of profound fatigue, neither after the ingestion of large amounts of fluid or of stimulants, as tea, coffee or alcohol. An overheated or unduly chilled extremity

will affect the arterial pressure in the part (see page 145). Observations made under pathologic conditions, such as edema or spasms, are absolutely unreliable.

Fear and psychic disturbances markedly influence the readings (see page 59); for this reason, in the nervous and excitable, the initial reading is often higher than those made subsequently.

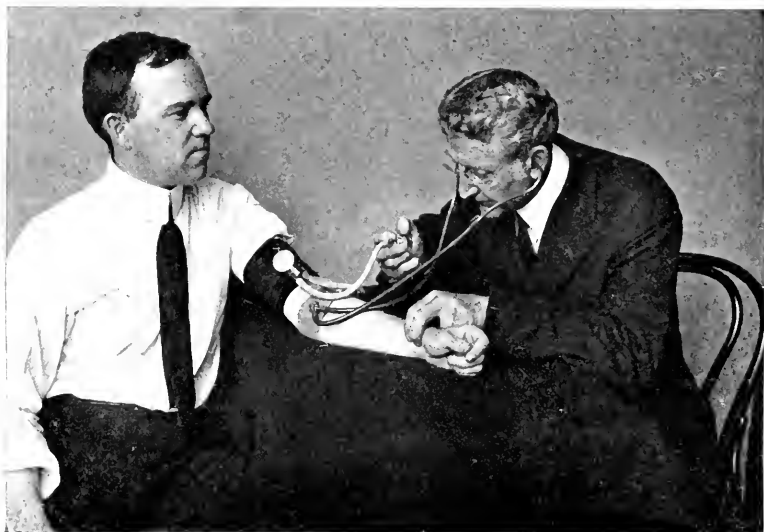


FIG. 6.—Auscultatory blood-pressure test.

Condition of Indicator and Cuff.—A leaky apparatus will give unreliable readings; old rubber parts are often responsible for this. When properly connected the apparatus should be able to sustain the mercury column without receding. A rapid fall indicates a leak somewhere in the air system and should be corrected. At the beginning of each test the indicator, irrespective of type, should register zero and in the mercury instruments the mercury

column should not be broken. This latter condition may be overcome by abruptly jarring the apparatus until the mercury unites.

The Performance of the Test.—The greatest rapidity compatible with accuracy is essential, since undue delay while the arm is under compression will, through vasomotor influences, give a disagreeable sensation, and may also affect the systolic pressure. Two or more readings should be made whenever possible for purposes of verification and to eliminate psychic and other transitory sources of variation, and no single observation should be accepted when it is possible to make additional ones.

		No.	
BLOOD-PRESSURE RECORD			
Name.....	Age.....	Sex.....	
Diagnosis.....	Date.....		
Systolic.....	m m.....	m m.....	
Diastolic.....	m m.....	m m.....	
Pulse Pressure.....	m m.....	m m.....	
Mean.....	m m.....	m m.....	
Pulse Rate.....	Reg. Irreg. Intermitt.		
TREATMENT.....			
REMARKS.....			

FIG. 7.—Author's index card.

The Keeping of Records.—Whenever possible blood-pressure records should be preserved, this not only makes for accuracy in individual readings but also furnishes valuable data for comparison, not only in the same case, but also in statistical studies.

For this purpose the author has for some time employed a small card upon which individual daily records are made and filed (see Fig. 7) and from which, when desired, graphic charts, similar to those shown throughout this work, are easily compiled.

Imparting Information to the Patient.—Except in rare instances the patient should not be given specific information regarding his blood-pressure, as it is extremely easy for certain individuals to become so imbued with the importance or omnipotence of this observation, that they develop a distinct neurasthenic stripe. There are, however, certain types of cases in which much good will follow a frank discussion of this subject with the patient, as for example, in the case of one who is firmly of the opinion that the height of his blood-pressure indicates a grave and rapidly fatal condition and who may be relieved permanently of this anxiety by demonstrating a physiologic level in the absence of such a condition. The other type is the neurasthenic who has inordinate reliance in this test and who if he can be shown a distinct improvement in pressure during the physician's conduct of his case, will naturally be helped to a recovery.

The Personal Equation of the Examiner.—Fortunately, for clinical medicine this factor is largely eliminated by the auscultatory method and now really exists only in determining the diastolic point according to the present interpretation of the sound phases (see Chapter V, page 90).

In the tactile method there is often an element of error of from 5 to 10 mm. between different observers. This may be due either to lack of experience and hence untrained tactile acuity, or to subjective pulsations felt by the observer. The frequency with which this subjective pulse becomes evident cannot be determined, but if suspected it can easily be eliminated by a simultaneous observation of the pulse of both the operator and the patient.

In my experience with students employing the auscultatory method there is rarely any appreciable element of error after the students have once learned to appreciate the significance of the sounds.

By tactile sense the determination of the diastolic pressure cannot be relied upon except when determined by those of constant and wide experience. Another source of error will depend upon whether the readings are made as the pressure in the instrument is ascending or descending (see page 141). It will be found here that, owing often to slight obstruction to the free passage of air in the apparatus, the pressure in the manometer will not immediately respond to the changes of pressure in the cuff. Another element of error may arise from the fact that, when using a pump or bellows, the rising pressure is not as easily controlled as the descending. Therefore, except under special conditions, all observations should be made after first rapidly and completely obliterating the pulse. There is one exception to this which has recently been called to my attention by Dr. Francis J. Dever, namely, in aortic regurgitation. Here in determining the diastolic pressure during the release the change from the third to the fourth phase may be so slight that it is missed, whereas with a rising pressure, after the diastolic pressure has been approximately determined, it will be found that the passage from the fourth to the third phase will be very sharp and distinct.

CHAPTER IV

THE SPHYGMOMANOMETER

METHOD OF ITS USE AND DESCRIPTION OF INSTRUMENTS

The use of manometers or upright tubes filled with fluid, in the study and measurement of blood-pressure in man is attributed to an English clergyman, Stephen Hales,¹ who published the results of his experiments in 1733, but it was not until recently that a systematic study of the circulation as shown by the blood-pressure was undertaken.

With our increasing knowledge of the circulatory mechanism, both in health and disease, it has become increasingly essential to have instruments of precision which will enable physicians to study circulatory phenomena, estimate circulatory efficiency and measure the work of the heart.

With the earlier instruments and the former methods only the systolic pressure could be gauged; but this is no longer sufficient, as the systolic pressure by itself may fail to give the information required. Both the systolic and the diastolic readings are now determined and in addition we have also learned to depend much upon other collateral data, coincidentally acquired with modern instruments and improved methods.

A great variety of apparatus for the estimation of human blood-pressure has been devised, the development and application of which having now become almost

¹Stephen Hales, "Statistical Essays," London, 1733, Vol. ii.

“ancient history,” they will be just touched upon here. In many of these older instruments either the principal or the technique or both were at fault; hence consideration need be given only to a few of the representative modern instruments of the last few years. These are great improvements over the previous ones, and have demonstrated their ability to give dependable and accurate blood-pressure values.

DEVELOPMENT OF THE SPHYGMOMANOMETER

Graphic methods for determining blood-pressure in man began with Vierordt in 1855 who attempted to measure blood-pressure by placing weights on the radial artery until the pulse was obliterated.

The first practical instrument, however, was introduced by Marey in 1876, but his work seems to have been practically forgotten until von Basch, in 1887 devised an instrument which at that time found some favor. This instrument recorded only the systolic pressure, and is important chiefly because it was the direct forerunner of all modern instruments. The instrument of von Basch employed intermediate compression—a rubber bulb filled with water and communicating by a tube with a mercury manometer, by which the radial artery was compressed. The systolic pressure was determined by measuring the amount of pressure necessary to obliterate the lumen of the artery and obstruct the pulse, which was indicated by the mercury column. In a later model von Basch substituted a spring manometer for the mercury column. In 1889 Potain replaced the water of the earlier instruments with air, and modified the pressure in the circuit at will

by means of a bulb, connected with the apparatus by a branch tube. He also improved his apparatus by using the chamber of an aneroid barometer for measuring pressure.

In 1896 Riva-Rocci¹ and Hill² published almost simultaneously articles descriptive of new sphygmomanometers. The important feature of each of these instruments was the introduction of a rubber bag or tube encircling the arm and inflated by a bulb or a pump.³ This improvement surmounted the most serious defect in the earlier instruments, which was the difficulty of accurately adapting the small round pelote to the arm, thereby compressing the artery (the radial) directly over the bone. By the method of Riva-Rocci and Hill, the pressure is everywhere exerted at right angles to the tangent of the circumference of the arm, and the artery therefore compressed equally from three sides against the bone (see page 66, Fig. 8).

Since this time there has been practically no change in the principles of sphygmomanometry. Improvements have tended toward perfecting the apparatus and simplifying the technique, changes having been directed chiefly toward portability, in means of circular compression and source of pressure, and the style of the manometer.

From the narrow arm-band as originally employed by Riva-Rocci (4.5 cm.—2 in.) to the extremely wide band of von Recklinghausen, numerous investigators have determined that a cuff 11 to 13 cm. (4½–5 in.) in width

¹ *Gaz. Med. di Torino*, 1896, Nos. 50 and 51.

² *Brit. Med. Jour.*, 1897, Vol. ii, p. 904.

³ For the accuracy of this method as compared with the direct or canular method, see page 39.

gives the most accurate readings, with the possible exception of the extremely obese. A special narrow cuff may also be found advantageous in work with very young children, manufactured at my suggestion by the G. P. Pilling and Son Company, Philadelphia, Pa.

Technique and Employment of the Sphygmomanometer.

—From the practical clinical standpoint all modern sphygmomanometers are applied and measure blood-pressure in the same manner. Therefore, a general description will explain the manner of application of all. For convenience of description the modern sphygmomanometer may be divided into the following parts:

1. The source of air pressure.
2. The compression device.
3. The manometer or indicator.

1. The *source of air pressure* may be a compressed air supply similar to that employed to operate atomizers, a rubber bulb, or a small metal force pump. The function of this portion of the instrument is merely to inflate the compression member and by so doing to exert a variable degree of pressure upon the artery under examination. The source of pressure is joined by suitable tubular means to the compression member.

2. The *compression member* is composed of a hollow elastic air-containing portion and a rigid, leather or fabric-retaining portion. These are so arranged that when applied to an extremity, the hollow elastic portion is in contact with the skin surface of the arm or leg, which it encircles either partially or completely. Outside this the restraining portion is fastened, in a suitable manner,

to restrain the elastic portion and to force the pressure inward against the member and so through it to the artery.

Fig. 8, *A* and *B*, shows the relation of the compression bag to the artery. In Fig. *A*, the pressure within the cuff

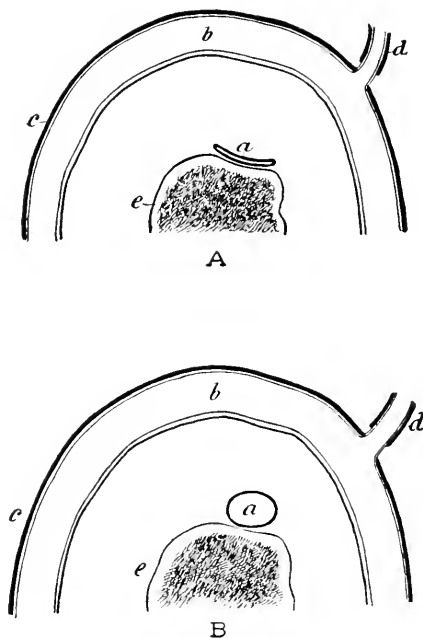


FIG. 8.—Diagram of relations of armlet to brachial artery. *A*. Pressure in "*b*" 135 mm. Hg., pressure in "*a*" 130 mm. Hg., *B* is therefore collapsed, pulse cannot pass. *B*. Pressure "*b*" 129 mm. Hg., pressure in "*a*" 130 mm. Hg., pulse passes. Explanation of systolic reading: *a*, artery; *b*, compressing armlet; *c*, retaining cuff; *d*, tube to manometer; *e*, humerus.

is greater than the blood-pressure within the artery, which is therefore collapsed and the pulse in the distal end of the vessel cut off. In Fig. *B*, the pressure in the cuff has been reduced so that it is a fraction of a millimeter less than the systolic pressure within the vessel. Now at

each systole, a small amount of blood will pass beyond the constriction and will reach the distal end of the artery, where the wave can be felt by the palpating finger at the wrist.

Fig. 9, A and B, represents the conditions existing

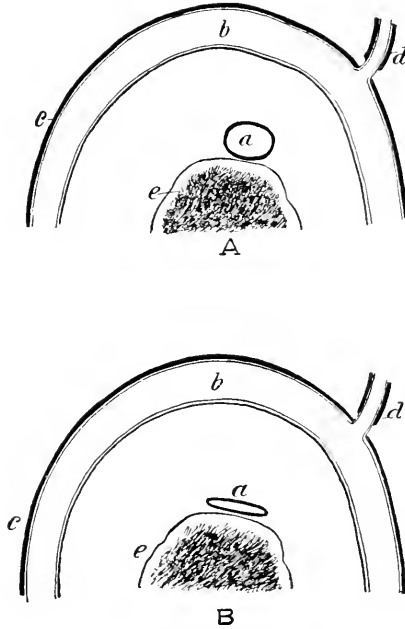


FIG. 9.—A. Systolic pressure in “a” 130 mm. Hg., pressure in “b” 101 mm. Hg., artery not compressed. B. Diastolic pressure in “a” 100 mm. Hg., pressure in “b” 101 mm. Hg., artery collapsed. Diagram of relation of armlet to brachial artery. Explanation of diastolic reading; *a*, artery; *b*, compressing armlet; *c*, retaining cuff; *d*, tube to manometer; *e*, humerus.

between the constricting cuff and the vessel at the diastolic time of pressure. A represents a pressure within the cuff less than the systolic pressure in the vessel. This is insufficient to affect the vessel during the systolic period, while B shows the artery and cuff during the diastolic

period, at the moment when the pressure within the artery is at its lowest point, a fraction of a millimeter less than the pressure within the cuff. Consequently the artery is collapsed at this time. The effect of each succeeding systole is to alternate between a round and a flat vessel at the point of compression. This affects the pressure of air within the cuff which is in turn transmitted to the mercury column of the manometer and becomes visible in the rhythmic fluctuation of the column of mercury which is synchronous with the pulse beat.

3. The *indicator, manometer or gauge*, is a device for measuring the amount of air pressure exerted against the artery. The modern types give the measure of pressure in terms of millimeter Hg., rendering the readings of all instruments comparable. The indicator is the part of the modern sphygmomanometer, which has had focussed upon its construction the greatest inventive skill and ingenuity. Many medical men of mechanical ability have made slight changes in minor or superficial details; as in shape and size of the manometer tube, or in the location of attachments for tubular connections in the mercury instruments, and in the form of the containing case in the aneroids. Hence we find a multiplicity of names attached to many slightly varying instruments.

Blood-pressure instruments are usually classified according to the style and type of measuring device employed. These may be grouped as follows:

CLASSIFICATION OF BLOOD-PRESSURE INSTRUMENTS

Modified from G. W. Norris, "Blood-pressure, its Clinical Applications," Lea and Febiger, 1914.

I. Mercury manometers:

1. Reservoir, Riva-Rocci, Cooke, Stanton, Pilling-Special and Pilling-Midget, Nicholson, Hill, Kercher, Hollman, Gärtner, Westenrijk.
2. U-shaped: Janeway, Faught, Martin, Linnell, Fellner.

II. Compressed-air manometers: Oliver, Benedick, Herz.**III. Aneroid manometers:** Rogers, Brunton, Faught, Pachon, Jacquet.**IV. Spring manometers:** von Recklinghausen, Stern.**V. Instruments for graphic registration:** Erlanger, Gibson, Bingel, Singer, Uskoff, Silbermann, Brugsch, Stursberg, Muenzer, Strauss-Fleischer, Bussenius, Wybauw**VI. Instruments constructed with, or to which may be fitted, special oscillating indices, such as the Fedde or Pal oscillometers, Bing, Faught, Vaquez, Widmer, Rodgers.****DESCRIPTIONS OF MODERN INSTRUMENTS**

1. Riva-Rocci Sphygmomanometer (Fig. 10).—This mercury manometer is of cistern form, and is constructed of heavy glass from which emerge two tubes, one for the inflating apparatus and one for the attachment of the arm-band. The latter is provided with a release valve for gradually lowering the pressure in the circuit during the test. The scale engraved upon the glass tube reads up to 260 mm. Hg.

The armlet consists of a hollow rubber tube covered with silk having a width of 4.5 cm. (2 in.), which is fastened

to the arm with a special clamp. The inflating apparatus is a double bulb such as is employed with a thermocautery.

Advantages.—The apparatus stands firmly on a solid base has a scale easily read, it is adjusted rapidly and is rapid in operation.

Disadvantages.—The armlet is too narrow for accurate readings, the apparatus is too fragile and totally unavailable for pressures over 260 mm. (10 in.) of mercury.

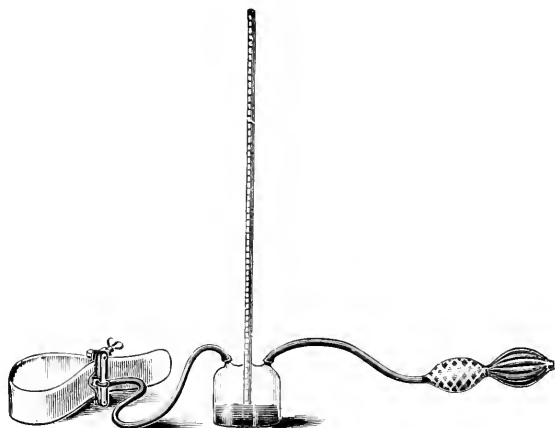


FIG. 10.—Riva-Rocci's sphygmomanometer. (Sahli and Potter.)

2. Cook's Modification of the Riva-Rocci (Fig. 11).—This is very similar to the preceding but is of lighter construction, and is provided with a jointed manometer tube which allows the instrument to be packed in a smaller space. It employs the double bulb inflator, and gives readings directly in millimeters Hg. which are read from a scale etched upon the glass.

Disadvantages.—It does not stand firmly being easily upset and broken. Without special care in packing and

transportation the mercury is frequently spilled. The caliber of the tube (1 mm.) is too small. The scale etched upon the glass is difficult to read.

3. Stanton's (Later Called Sands) Sphygmomanometer (Fig. 12).—This instrument was devised in an effort to increase portability, to reduce the probability of breakage

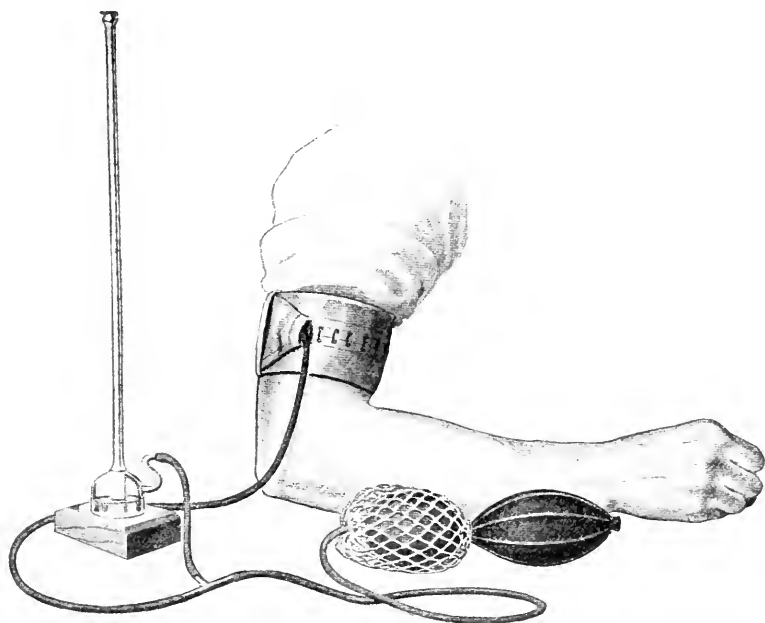


FIG. 11.—Cook's modification of the Riva-Rocci sphygmomanometer, showing narrow arm-band in place, with cautory bulb inflator.

and to lessen the elasticity of the tubular system, thereby improving the diastolic fluctuation of the mercury.

These changes were effected by substituting a metal cistern and by arranging over this a screw joint for the attachment of the vertical glass tube; also by the introduction of a stopcock in the short tube as it emerges from the cistern to serve for attachment of the inflating bulb.

This eliminates the elasticity of the inflating apparatus during the diastolic reading. The instrument employs the standard 12-cm. ($4\frac{1}{2}$ -in.) cuff, retained on the arm by a canvas outer cuff and buckle straps.

Disadvantages.—Chiefly the time and skill required to set up the apparatus before using, and the great difficulty

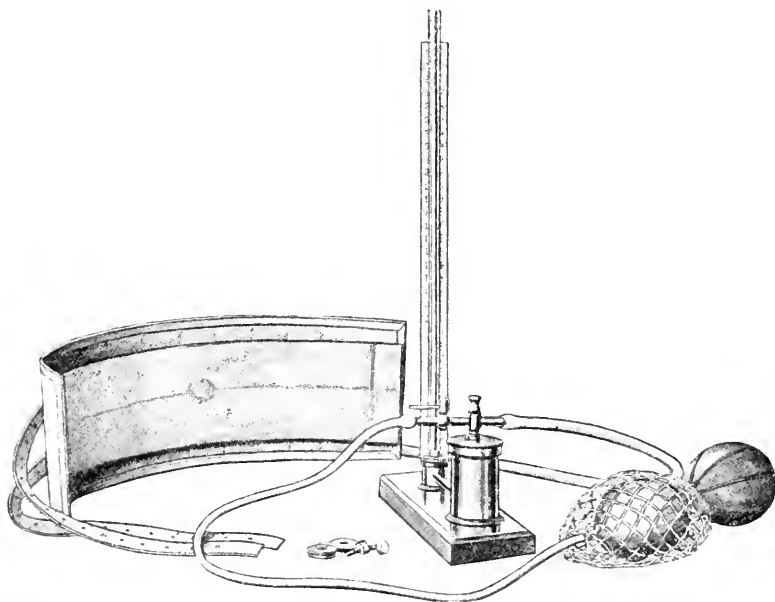


FIG. 12.—Stanton's sphygmomanometer, showing arrangement of parts with cautey bulb inflator.

in avoiding the loss of mercury during the setting-up process. Also the cistern arrangement gives low readings in high pressures.

4. Janeway's Sphygmomanometer (Fig. 13).—In the construction of the Janeway apparatus we see a return to the U-tube type, first devised by von Basch. This form appears to be a more accurate method of employing the

mercury column, as in the cistern form no cognizance is taken of the change in the level of the mercury in the cistern, which must, for physical reasons, give too low readings when employed in the study of high pressures.

This instrument employs the circular compression band of standard width and a Politzer bag for inflation. In

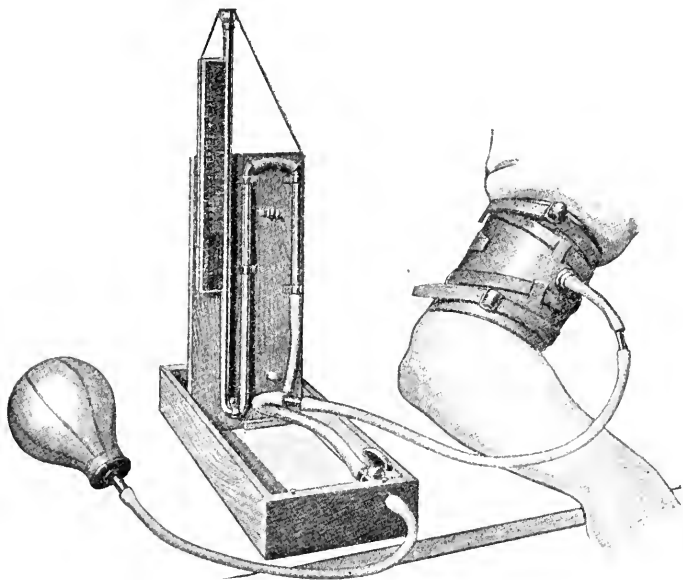


FIG. 13.—Janeway's sphygmomanometer, attached to arm, showing method of retention of cuff, arrangement of manometer, with Politzer bag inflator.

addition to this the only original feature of this instrument is the jointed U-tube which allows the instrument, with the exception of the cuff and inflating bag, to be contained in a case measuring $10\frac{1}{4} \times 4\frac{5}{8} \times 1\frac{7}{8}$ in. and weighing $2\frac{1}{2}$ lb. The open end of the manometer tube is closed with a cork when not in use, and the rubber connection on the other limb leading to the attachments is compressed by

closing the case, to prevent loss of mercury from the manometer. The scale is arranged to slide down into the box when not in use. The arm-band is 12 cm. ($4\frac{1}{2}$ in.) wide and is retained on the arm by a non-elastic outer cuff provided with friction straps.

Advantages.—This apparatus is light, compact and portable and the readings accurate.

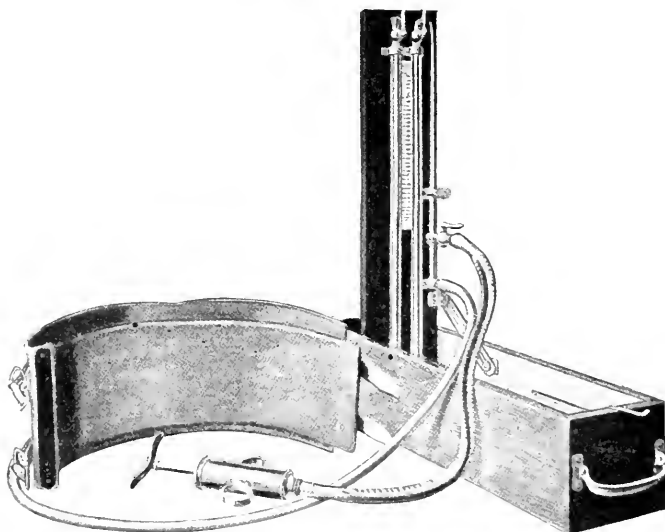


FIG. 14.—Faught's mercury sphygmomanometer, showing relation of parts, metal pump, and special expansion tubing for inflator.

Disadvantages.—The frailness of jointed U-tube. The mercury is easily spilled because of the inadequate method employed to confine it when the instrument is not in use. The rubber connections are short-lived and must often be renewed.

5. The Faught Standard Mercury Sphygmomanometer (See Fig. 14).—This apparatus was devised early in 1909, in an endeavor to overcome the shortcomings of existing

instruments, the majority of which were found to be frail, besides requiring special skill to operate, consuming too much time and being above all defective mechanically.

The Standard sphygmomanometer is of the U-tube type and all complicated parts have been either simplified or eliminated. The complete apparatus including the cuff of standard width, and the inflating pump may be enclosed in a mahogany carrying case, measuring $14 \times 4 \times 4\frac{1}{2}$ in. The lid is hinged and locks in a vertical position to serve as the support of the manometer and the connections. Each arm of the U-tube is provided with a guard cock which remains closed, except during actual use of the instrument, thus preventing absolutely any loss of mercury, excepting with gross carelessness. There are no rubber connections to wear out. The upper nipple to which the pump is attached is provided with a stopcock, which must be closed during the systolic and diastolic readings. The millimeter scale, which can be adjusted to the level of the mercury is reduced one-half to compensate for the fall of the mercury in the other limb, thus the markings give the reading directly in millimeters of mercury. The pressure is obtained by the use of a metallic pump attached to the upper nipple by means of a collapsible rubber tube of special construction, which by its expansion during the operation of the pump, reduces the impact of air before it reaches the mercury in the manometer, and takes the place of the second bulb of the cautery apparatus. An escape valve is provided for gradually lowering pressure during the test.

Disadvantages.—It being perhaps somewhat difficult for one to see the defects in an apparatus of his own

devising, criticisms of this instrument are not here attempted, but are left to others. It is believed that the size of the Standard, and its weight as compared with the more recently devised pocket types of sphygmomanometer, should not be considered detrimental, as many physicians prefer the mercury type, and use it exclusively in physiologic research.

6. The Folding Mercury Sphygmomanometer.—The folding mercury instruments of Cooke and of Janeway, already described, are not at present in great favor, because of the sectional glass tube, which favors the loss of mercury and which is so fragile. These defects have been largely overcome by two surgical instrument makers who, almost simultaneously presented perfected instruments which are in appearance quite similar to each other. One is called the Midget¹ (see Fig. 15) and the other known as the Nicholson.² These instruments follow the design of the Riva-Rocci apparatus, in that they employ the reservoir and vertical-tube manometer. In the Midget instrument, with which I am more familiar, the important point is the improved construction in the hinge which serves to join the two segments of the manometer tube, so arranged that the old cone-socket ground-joint connection is abolished, so that, when in position for operation, the two ends of the tube are in close contact, being separated only by a thin washer which prevents the escape of mercury. This arrangement not only reduces the danger of breakage to a minimum but also avoids obstructing

¹ Manufactured by George P. Pilling & Son Co., Philadelphia.

² Manufactured by Precision Thermometer and Instrument Co., Philadelphia.

the view of the mercury column at any point on the scale, a highly desirable condition which is not met by earlier jointed instruments. The apparatus is placed in the operative position simply by opening the lid which locks when vertical. The tube with its scale is then unfolded and pressed vertically upward until a spring-catch maintains it

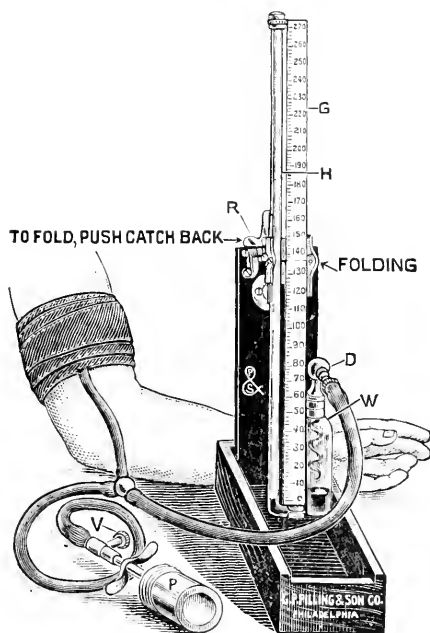


FIG. 15.—Folding mercury sphygmomanometer in operative position, showing details of construction and attachment to arm.

in this position. After use a lever arrangement releases this lock and permits of folding, which movement automatically closes the mouth of the proximal tube. The scale, while being a separate unit and adjustable vertically to accommodate changes in the mercury level, nevertheless follows the manometer tube in all positions. The apparatus

registers up to 260 mm. and when folded is self-contained in a mahogany case measuring $7 \times 2\frac{3}{8} \times 1\frac{5}{8}$. The apparatus employs the standard flexible cuff and is inflated by a small hand-pump.

7. The Kercher Mercury Sphygmomanometer.—This little instrument is another modification of the cistern-tube type of apparatus and was suggested in 1910 by Delno E. Kercher,¹ is supported on a wooden base and is composed of a small glass cistern containing the mercury and an upright manometer tube reading to 300 mm. The instrument is provided with a pneumatic cuff of 4×36 in. and is actuated by a rubber bulb with double valve, provided with a T-connection for attachment of the arm-band and manometer.

8. The Aneroid Sphygmomanometer.—This type of apparatus has much to recommend it and should, if the physician is supplied with an accurate model, be most satisfactory for all purposes, as the present methods of pressure reading do not depend upon the oscillation of the mercury column.

The term aneroid is a misnomer and should never have been applied, but, as it has become so firmly associated with this apparatus, it probably will remain. Aneroid signifies the absence of air, *i.e.*, the presence of a vacuum, yet there is no vacuum chamber in this sphygmomanometer, or in any as far as I know.

The vital portion of these instruments, or the part which actuates the indicator, consists of a group of metallic chambers, made of a special alloy of paper thinness, which when exposed appears not unlike the aneroid portion of the well-known barometer.

Concerning the mechanism of this type of sphygmo-

¹ *Jour. A. M. A.*, Apr. 2, 1910, Vol. liv, 14, p. 1136.

manometer close examination reveals radical differences in the mechanism of instruments belonging to this class. References to Figs. 16 and 17 show diagrammatically

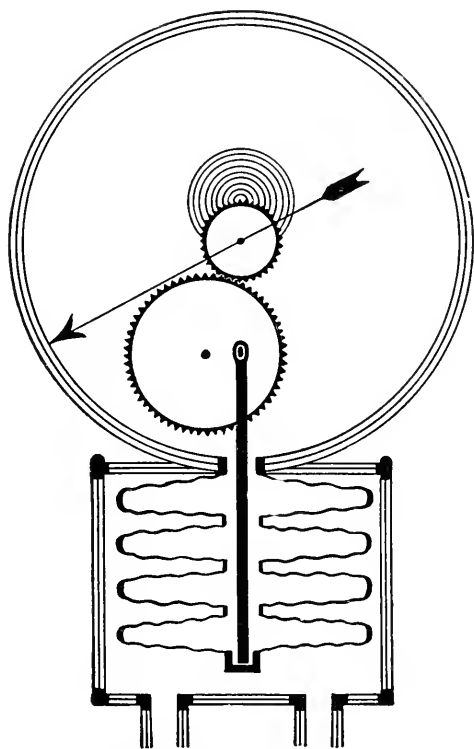


FIG. 16.—Diagrammatic representation of internal construction of Faught pocket sphygmomanometer. Note the four compression chambers which are intercommunicating one with the other and with the atmosphere through the body of the instrument; also the two nipples, through which the compressing force is directed upon the outside of the chambers causing their collapse, thereby actuating the vertical rod which extending upward connects with the gears as shown.

this difference. Fig. 16 which is an enlarged transverse section of a Faught pocket sphygmomanometer as compared with Fig. 17 shows that in the author's instrument,

the indicator is actuated through two gear-wheels, which are in turn operated by a plunger which is seen to pass through and be attached to the bottom of the lowest chamber. It

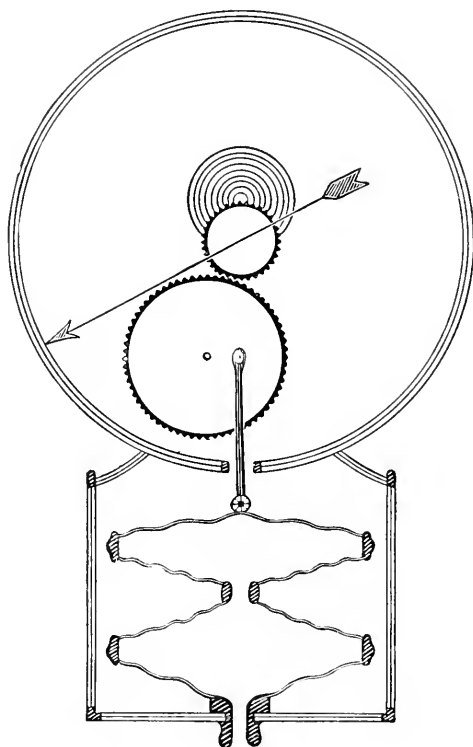


FIG. 17.—Diagrammatic representation of internal construction of a well-known pocket sphygmomanometer. Note the two compression chambers, the single nipple and the arrangement whereby the compressing air is directed within, and expands the chambers.

will be seen also that these chambers communicate with each other and with the air in the body of the apparatus. The two attachments which serve as connections for the pump and the arm-band are seen to be interchangeable and

to communicate with a closed space which surrounds, but which does not communicate with, the interior of the chambers. This is the vital point which assures accuracy in this instrument for, when air pressure is increased around the chambers, they tend to become collapsed and, driving the plunger upward, operate the indicator through the gears. The amount of movement depends upon the amount of pressure exerted upon the chambers, of which there are *four*.

Reference to Fig. 17 will show that the principle here is the reverse, and that the *two* chambers are designed to receive the increment of air, which, by expanding them, operates the plunger attached to their upper face which in turn actuates the indicator through a series of gears. In this instrument the over-expansion of the chambers is a constant possibility which, occurring even once, renders the apparatus permanently incorrect, while in the Faught the pressure can only collapse the chambers which, even when over-compressed, cannot more than force them into contact, and as this manipulation is applied to every Faught apparatus before it is graduated, the danger of inaccuracy is entirely removed. Another point in favor of this instrument is the employment of four chambers instead of two; this greatly reduces molecular motion and is another element in reducing the possibility of inaccuracy.

9. Faught pocket sphygmomanometer (Fig. 18) has a dial of white enamel, somewhat similar to that of a watch. The scale is graduated in millimeters of mercury, as determined by accurate calibration with a standard mercury column. The numerals are in red and black, to facilitate reading, and each individual graduation represents 2 mm.,

giving a working scale extending from zero to 300. No mathematical calculations are necessary to compute the pressure, which can be easily read directly from the dial.

In order to insure accurate and unvariable readings at all points on the scale, a factor of safety of 150 mm. has been provided, *i.e.*, each Faught pocket sphygmomanometer before leaving the factory is tested up to 150 mm. above the

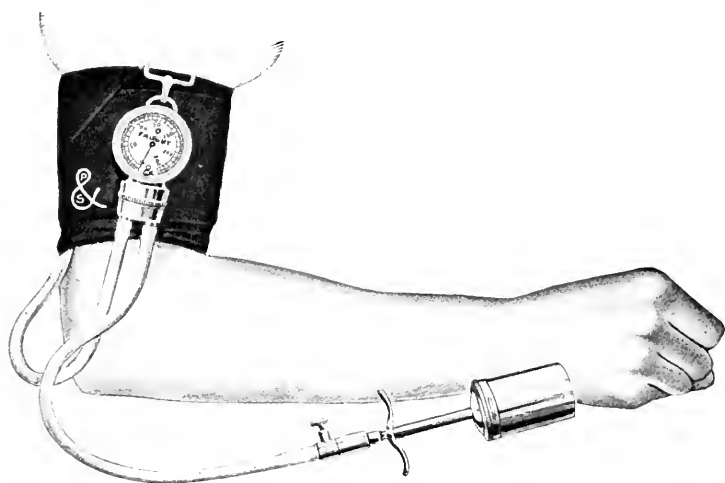


FIG. 18.—Faught pocket sphygmomanometer attached to arm showing position of arm-band, dial attached to hook and arrangement of tube connections.

300 on the scale, or to 450, after which the readings must correspond with those of a standard mercury column, and the needle after this severe test must return immediately to zero. This shows clearly that, with ordinary use, it is practically impossible to distort the compression chambers of the instrument.

The accuracy of the so-called aneroid, or diaphragm type of sphygmomanometer has been questioned by some,

but as already stated in the improved type of this instrument sudden variations are very unlikely to occur. The so-called "fatigue of metal" referred to by some authorities does not exist, and any error which manufacturers admit might develop in their instruments, must be due to some mechanical defect.

An additional member of the so-called aneroid group is the "clinical" pocket sphygmomanometer which has been placed on the market at the author's suggestion. This instrument is shown in Fig. 20. It is identical in construction to the pocket instrument differing only in having a specially large dial ($3\frac{1}{2}$ in.) and consequently, a more easily read scale, reading to 350 mm. Hg. The markings on this scale are sufficiently distinct to be easily read at a distance

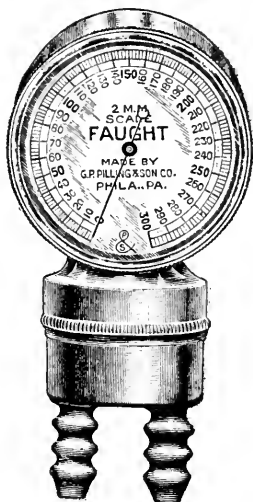


FIG. 19.—Faught pocket apparatus dial in detail.

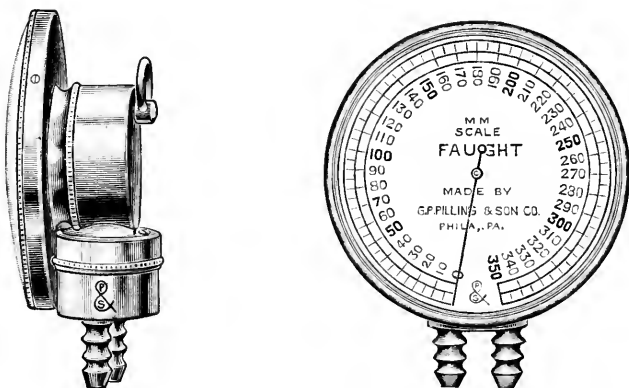


Fig. 20.—Clinical sphygmomanometer; side and front views; $\frac{1}{2}$ actual size.

of 10 to 15 ft., an advantage that will recommend itself to class teaching.

The instrument employs the flexible bandage-cuff or arm-band, (see Fig. 18), the inflatable portion of which measures 5×9 in. A small metal pump with exhaust valve attached is supplied and these parts are all contained in a Morocco pocket case. The needle of the indicator is extremely delicate and so sensitive that a diastolic reading can be made in any case in which a mercury

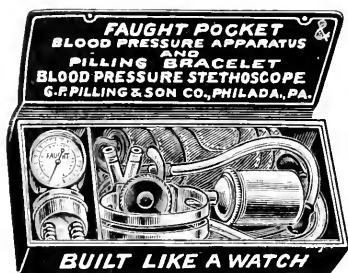


FIG. 21.—Faught pocket sphygmomanometer in case.

10. Erlanger's Sphygmomanometer (Fig. 22).—This instrument in its improved form is apparently the most accurate yet devised for determining blood-pressure, being based upon the same principle as the other instruments, but both the return of the pulse and the point of maximum pulsation are made clearly visible, thus almost entirely removing subjective errors.

The construction of this instrument is much more complicated though the only essential difference is the addition of an original recording device. The U-tube manometer connects with a four-way tube, of which one branch leads to the armlet, and another to a special stopcock. The vertical branch communicates with the interior of a rubber bulb, enclosed within a heavy glass bulb, which in turn, under certain conditions communicates freely with the atmosphere through another tube returning to the special

stopcock. The object of this glass-encased rubber bulb is to shield the delicate tambour from too sudden changes in pressure. The tambour communicates with the air in the glass bulb outside of the rubber ball, and operating an aluminum needle above the tambour, inscribes its movements on a revolving drum. This makes a tracing upon smoked paper as in the ordinary kymographion. The whole is

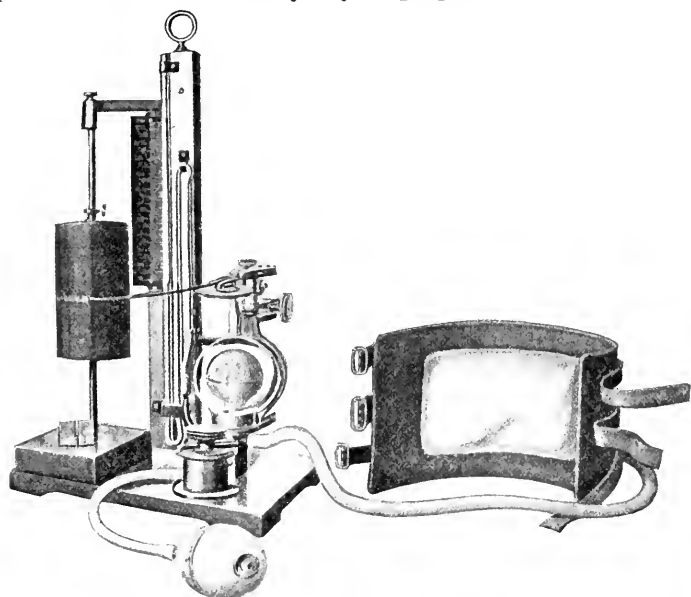


FIG. 22.—Erlanger's sphygmomanometer with kymographion in place, showing arm-band and atomizer-bulb inflator.

attached to a metal base and is covered for transportation by a metal case which is somewhat larger than a microscope box and of about the same weight.

The standard cuff is employed and pressure is obtained from a Politzer bag. All rubber tubing is of the high-pressure variety to afford rigidity.

The minute details of construction and the operation of

the special stopcock are too extensive to include here, suffice to say that with practice in handling the instrument the readings obtained are accurate and furnish a permanent graphic record of both systolic and diastolic pressures.

Disadvantages.—The chief fault to be found with this apparatus is from the standpoint of clinical availability. Its bulk and weight render it almost useless for clinical work except perhaps in the office or the hospital. The technique of smoking the cylinder and of making necessary adjustments consume more time than can generally be spared during the activities of an extended practice. And in addition the rubber connections and the diaphragm of the tambour often need replacing at most inconvenient times.

The value of graphic records to-day is sufficiently obvious and needs no argument. We would know little of the characteristic temperature curves of malaria or typhoid fever if we depended for our information upon a long column of figures. The course of blood-pressure is equally easy to chart and the curve thus obtained tells us at a glance much that the perusal of the usual written record would fail to convey. In both acute and chronic diseases and during operations the systolic blood-pressure and the pulse should be charted at regular intervals.

This chart is arranged in the form of a combined pulse, temperature and blood-pressure chart, the several scales being so placed that the pulse, temperature and blood-pressure curves do not become superimposed (see Fig. 23).

The chart sheet measures 9×12 in., which is the size of the usual hospital history sheet. The chart may be filled in, in different colors if desired to make the record more graphic, but this is not necessary to its accurate keeping.

These charts can be obtained in pads of twenty-five from any surgical instrument dealer at a nominal price.

PULSE, TEMPERATURE AND BLOOD PRESSURE CHART

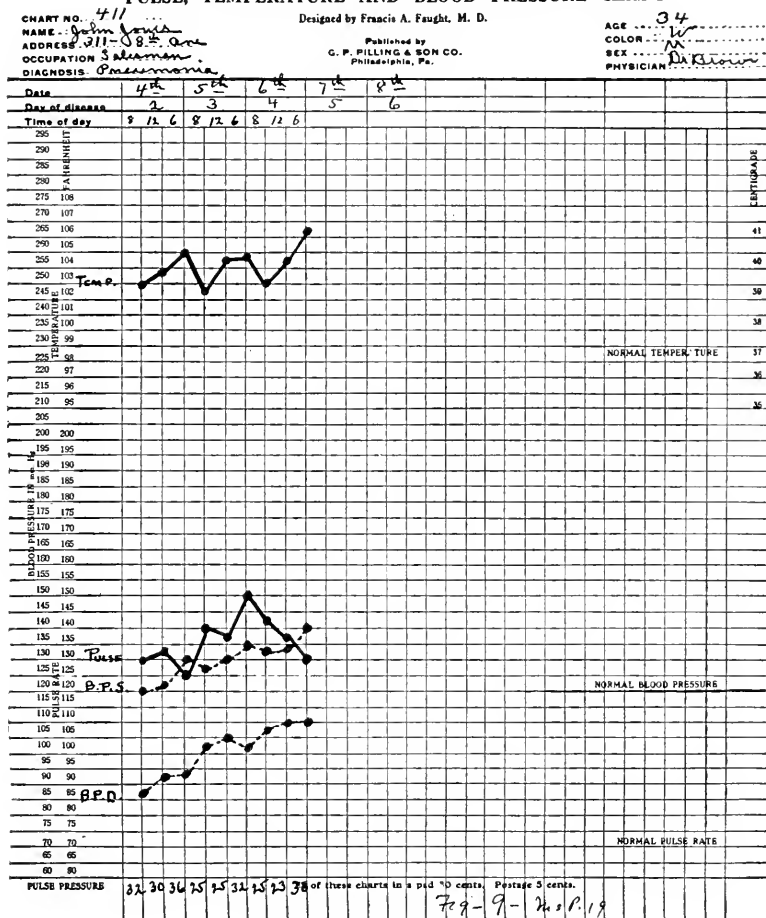


FIG. 23.—Specimen chart.

11. The White Mercury Sphygmomanometer (Fig. 24).

—The special sphygmomanometer devised by White is particularly designed to meet the requirements of colleges

and schools, where the demonstration of blood-pressure phenomena is made before a large body of persons. This instrument contains some of the elements of the Faught mercury sphygmomanometer, to which have been added an electrical signal device as shown in Fig. 24. By adjusting

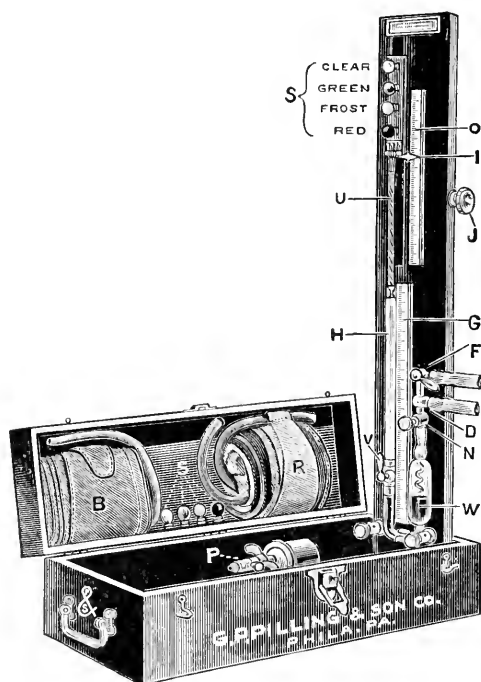


FIG. 24.—Pilling-White Graphic Sphygmomanometer. Designed to demonstrate by electrical contacts actuating colored lamps the excursions of the pulse pressure.

the lamp carriage to the proper position, both the systole and the diastole of the pulse is shown by miniature electrical bulbs. By having the lights of different colors the demonstration is even more graphic and the extent of the oscillation of the pulse wave is shown by the number of lamps illuminated during each cardiac impulse.

CHAPTER V

THE CLINICAL DETERMINATION OF BLOOD-PRESSURE

The Scope of the Test.—The clinical determination of blood-pressure involves an estimation of the systolic and the diastolic pressures, from which may be determined the pulse pressure and the mean pressure. The value of the observation is enhanced if the pulse rate is recorded as a part of this examination.

Any peculiarities noted either while palpating the pulse or in variations from normal in the series of sounds heard during auscultation of the vessel should be recorded. Valuable supplementary information may be developed through a careful consideration of these factors.

For convenience in recording blood-pressure observations the author has devised and now employs the printed card index form shown in Fig. 7, page 59.

Apart from the style of the apparatus there are several methods from which to choose for determining blood-pressure. While any may be employed, there is considerable difference in their accuracy and applicability. It seems advisable when there is choice, to employ that one which has been most amply shown by clinical and experimental evidence to be the most accurate and satisfactory, *i.e.*, the auscultatory. The several methods which may be employed are the following:

The auscultatory.

The palpatory.

The visual.

The graphic.

The oscillatory.

A detailed description of each together with its advantages and disadvantages follows.

A. THE AUSCULTATORY METHOD

In 1905 Korotkoff¹ reported that when the bell of a stethoscope was placed over the brachial artery at its bifurcation just below the cuff of a sphygmomanometer

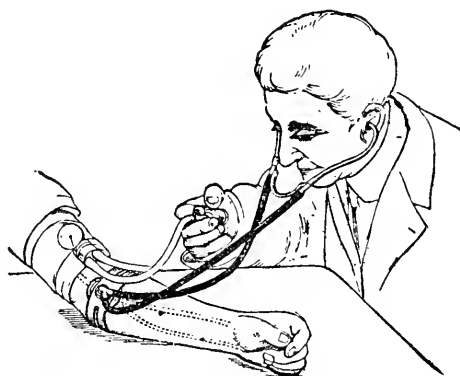


FIG. 25.—Showing relation of sphygmomanometer and bracelet stethoscope arranged for the auscultatory blood-pressure test.

(see Fig. 25) and the pressure in the cuff gradually reduced, a series of characteristic sounds were heard. He described three tone phases and stated that they bore a definite relation to the character of the pulse, the condition of the heart and to the systolic and the diastolic blood-pressure. Subsequent observers, among them Ettinger,² Lang and

¹ *Mitt. d. k. k. mil. Med. Akad. zu St. Petersburg.*, 1905, xi, 365.

² *Wien. klin. Wochen.*, 1907, xx, 992.

Manswetowa,¹ Fisher,² Gittings,³ Goodman and Howell,⁴ and Swan⁵ have further analyzed these sounds and have shown that the three tone phases of Korotokoff may usually be separated into five.

The operator, in employing this method notes the point, on the millimeter scale, either the mercury column or the dial indicator, of appearance of (a) the first tap, (b) the appearance of the systolic murmur, (c) the disappearance of the systolic murmur, (d) the point at which the sound suddenly diminishes in intensity and (e) the point at which the sound ceases. These several criteria determine the five phases of Ettinger. Korotokoff's original analysis of these auscultatory arterial sounds distinguished only three—the first or systolic point corresponding to the first tap, the second to the development of the systolic murmur, and the third to the disappearance of all sound.

Swan in his recent and most exhaustive treatment of this subject⁶ calls attention to the confusion existing in literature concerning the phases. This, he says, is due to the fact that some authors use the term *phase* to indicate the *points* at which the various changes in the character of the sounds occur, while other writers speak of the period between these two points as a *phase*. Thus the early writers call the point at which the first sound is heard the first phase; that at which the murmur appears as the second phase; that at which the murmur disappears as the third phase;

¹ *Deutsch. klin. Med.*, 1908, xciv, 445.

² *Zeitsch. f. diätet. u. physick. Therap.*, 1909, xii, p. 389.

³ J. C. Gittings, *Arch. Int. Med.*, August, 1910, p. 196.

⁴ *Am. Jour. Med. Sci.*, 1911, cxlii, p. 334.

⁵ *Internat. Clinics*, iv, Series, 24.

⁶ *Loc. cit.*

that at which the sound following the murmur becomes suddenly lessened in intensity the fourth phase; that at which the sound entirely disappears the fifth phase. On the other hand, recent writers call the duration of the first sound as measured in millimeters of mercury, the first phase; the duration of the murmur the second phase; the duration of the second clear sound the third phase; and the duration of the dull end sound the fourth phase. In order to avoid confusion Swan suggests the adoption of the word *point*, to indicate the changes in the sounds and the use of the term *phase* to indicate the duration of the sounds respectively.

The general adoption of this suggestion will contribute greatly to clarity in our discussions and writings upon the auscultatory method of study; which if accepted, will admit of the following classification:

First point: Appearance of first tap, or sound in previously silent stethoscope.

First phase: A sharp sound not unlike the cardiac first sound.

Second point: The instant of the addition of a systolic murmur to the first phase.

Second phase: The duration of the hissing murmur plus the first sound.

Third point: The moment of disappearance of the systolic murmur.

Third phase: The duration of a sound similar but somewhat more powerful than the first.

Fourth point: The moment that the sound of the third phase suddenly becomes muffled and reduced to a dull tone.

Fourth phase: The duration of the muffled soft tone.

Fifth point: All sound disappears.

The Significance of the Points and Phases.—*The first point*, which is the appearance of the first clear sound, has been accepted by all observers to indicate the systolic blood-pressure. Cook and Taussig¹ describe it as the “most accurate for determining this pressure. It is as much superior to the palpation of the radial as the sense of hearing is keener than the sense of touch.”

Warfield² has proved by experiments on dogs that the first phase corresponds to the systolic blood-pressure.

The fourth point which is the time at which the tap, heard after the disappearance of the systolic murmur, becomes dull, is considered by Land and Manswetowa³ Zabel,⁴ Taussig and Cook,⁵ Stone,⁶ Hirschfelder⁷ and Warfield⁸ to indicate the diastolic pressure.

The fifth point or the moment of disappearance of all sounds is, on the other hand, considered to indicate the diastolic pressure by Korotkoff,⁹ Ettinger,¹⁰ Gittings,¹¹ Goodman, and Howell,¹² Hooker and Southworth,¹³ Krylow,¹⁴

¹ *Arch. Int. Med.*, 1913, xi, 542.

² *Arch. Int. Med.*, 1912, x, 258 and *Interstate Med. Jour.*, 1912, xix, 856.

³ *Deutsch. Arch. f. klin. Med.*, 1908, xciv, 441, 455.

⁴ *Berl. klin. Wochen.*, 1909, xlvi, 1352.

⁵ *Arch. Int. Med.*, 1913, xi, 542.

⁶ *Jour. A. M. A.*, 1913, lxi, 1256

⁷ “Diseases of the Heart and Aorta,” edition 2, 1913, 33.

⁸ *Loc. cit.*

⁹ *Mitt. d. k. mil. med. Akad. zu. St. Petersburg*, 1905, ix, 365.

¹⁰ *Wien. klin. Wochen.*, 1909, Feb. 11.

¹¹ *Arch. Int. Med.*, 1910, vi, 195.

¹² *Univ. Penn. Med. Bull.*, 1910, xxiii, 469 and *Amer. Jour. Med. Sci.*, 1911, cxlii, 334.

¹³ *Arch. Int. Med.*, 1914, xiii, 384.

¹⁴ Quoted by Gittings and Goodman and Howell.

Fellner,¹ Fisher,² Ehret,³ Schrumpf and Zabel,⁴ Moritz,⁵ Sterzing,⁶ Prendergast⁷ and Bickel.⁸ Bickel,⁹ found that the fourth point corresponds to the diastolic pressure obtained by the graphic method and approximates the diastolic pressure as obtained by the palpatory method.

The fifth point, however, corresponds to the diastolic pressure as obtained by the oscillatory method. Zabel¹⁰ made his observation in a manner the reverse of that of other observers and while he obtained similar results, Swan¹¹ has noted important objections to making diastolic readings in this manner, the chief of which is that the adventitious sounds made by inflating the cuff obscure the sharp fifth point.

In a careful review of this work Swan concludes that, as conditions are not as ideal in clinical work as those found by Warfield in his experiments, as the cuff is applied through intervening tissues and because the fourth point is often absent in clinical work, it is advisable for bedside work to record the fifth point as the diastolic pressure.

The contention that the interpretation of the diastolic pressure as indicated by the fourth point makes an important difference in determining mean pressures and pulse

¹ *Verhandl. d. 24. Kong. f. inn. Med.*, 1909, xxiv, 404.

² *Deutsch. med. Wochen.*, 1908, xxxiv, 1141 and *Zeitsch. f. diätet. u. physik. Therap.*, 1909, xii, 389.

³ *München. med. Wochen.*, 1909, lvi, 959.

⁴ *München. med. Wochen.*, 1909, lvi, 704.

⁵ *München. med. Wochen.*, 1909, lvi, 321.

⁶ *Deutsch. med. Wochen.*, 1909, xxxv, 1874.

⁷ *N. Y. Med. Jour.*, 1913, xcvi, 78.

⁸ *Zeitsch. f. exper. Path. u. Therap.*, Bd. c. H, quoted by Zabel, *Berl. klin. Wochen.*, 1909, xlvi, 1352.

⁹ *Loc. cit.*

¹⁰ *Berl. klin. Wochen.*, 1909, xlvi, 1352.

¹¹ John M. Swan, *Inter. Clinics*, iv, Series 24.

pressures is quite well founded. Swan found that in 118 observations in which both the fourth point and the fifth point were recorded the average difference amounted to 14 mm.

Warfield found that the difference between the fourth and fifth points in some cases amounted to 12 mm., in other cases the difference amounted only to from 2 to 4 mm.

Analysis of the Tone Phases.—The *first sound* or *phase* is ascribed to the return of the pulse below the obstruction which suddenly distends the collapsed brachial artery. Gittings¹ believes that these sound waves are reinforced by the resonating quality of the cuff, since compression with an Esmarch bandage usually fails to produce this character of sound, although MacWilliams and Melvin² using a schematic circulation, were able to obtain the characteristic sounds when no cuff was used. They attribute the sounds to the "vibration of the arterial wall when the normal circular (cylindrical) form of the vessel is, in the compression area, more or less distorted by external pressure."

The Second Phase.—The addition of a murmurish quality to the first sound due to the relatively large size of the artery below the constriction which produces fluid-veins.

The Third Phase.—The disappearance of the murmurish quality of the second sound is probably the hardest to explain. It would appear to be due to the gradual lessening of the constricting pressure and the consequent elimination of the fluid-veins. This phase is usually very distinct and often louder than the first. It is the phase of the

¹ *Arch. Int. Med.*, August, 1910.

² *Heart*, 1914, v. 153.

greatest duration and corresponds to the time of greatest oscillation in the indicators and in the graphic apparatus.

The Fourth Phase.—A muffling of the sound which occurs when the pressure from the constricting band has become so slight that it fails to compress the artery sufficiently, even during the diastolic period, to either slow the blood-stream or produce fluid-veins (see Fig. 9,B, page 67).

The disappearance of all sounds occurs when normal arterial relations are reestablished.

In cases of aortic regurgitation the fourth phase is usually persistent, and it is not uncommon to hear a sharp sound over the arteries when no pressure is being exerted by the cuff. This phenomenon may occasionally be noted in other conditions and therefore cannot now be considered as pathognomonic of aortic regurgitation. The persistence of the fourth phase, or the observation of the fifth point at zero, has been observed in broncho-pneumonia and exophthalmic goiter¹ in high fever and cardiac neuroses;² in arteriosclerosis;³ in hypertrophic cirrhosis and hyperthyroidism⁴ while Cook and Taussig⁵ have found it at 10 mm. Hg. in a series of cases of aortic regurgitation.

Variations in Tone Phases and their Significance.—When auscultating for the determination of blood-pressure, not only may these variations be detected, but in addition slight arrhythmias and variations in the force of each systole. This fact is known to many. (The author called attention to this point in his ward-class teaching over six years ago).

¹ Cook and Taussig, *Arch. Int. Med.*, 1913, xi, 542.

² Fischer, *Deutsch. Med. Wochen.*, 1908, xxxiv, 1141.

³ Zabel, *loc. cit.*

⁴ Swan, *loc. cit.*

⁵ *Loc. cit.*

Quite recently it has again been brought forward by the work of Jas. B. Herrick.¹

First Phase.—It has been noted that a strong first phase is not always an indication of health, and Fischer² has noted the fact that in cases of very high pressure, as in chronic nephritis, the sounds of the first phase are unusually loud.

Second Phase.—Fischer³ and Goodman and Howell⁴ have found the murmurs of the second phase usually very clear and the phase itself prolonged in cases of anemia. Tornai⁵ states that a strong murmur phase indicates a good heart, and that a phase of increased length after exercise indicates a good heart, whereas if the murmur disappears after exercise it is an indication of a weakened left ventricle. A murmur phase which gradually increases, and gradually recedes—that is not clear cut at the ends—is noted in cases of dilatation and hypertrophy. The absence of this phase is an indication of heart weakness. Goodman and Howell,⁶ and Swan⁷ believe that the second phase is the first to suffer in failing compensation. They believe the length of this phase indicates cardiac efficiency, and they have worked out a cardiac efficiency ratio, which is based upon this belief (*vide infra*).

Third Phase.—Fischer⁸ considers the third phase the most important clinically. A long, loud and clear third

¹ *Jour. A. M. A.*, Feb. 26, 1915, lxiv, 9.

² *Loc. cit.*

³ *Loc. cit.*

⁴ *Am. Jour. Med. Sci.*, 1911, cxlii, 334.

⁵ *Zeitsch. f. diatet. u. physick. Therap.*, xiii.

⁶ *Loc. cit.*

⁷ *Loc. cit.*

⁸ *Loc. cit.*

phase, indicates cardiac strength, while a weak and short third phase with soft sounds is indicative of cardiac weakness. Ettinger, Warfield, Goodman and Howell, and Swan concur in this view.

Fourth Phase.—The duration of the fourth phase increases with cardiac weakness and according to Fischer¹ is almost always a concomitant of a low diastolic pressure.

The Cardiac Strength—Cardiac Weakness Ratio.—Basing their conclusions upon the preceding premises, Goodman and Howell in 1910² worked out a percentage duration of the pulse pressure for each tone phase, considering that the second and third phases are dependent upon cardiac strength or effectiveness, while the first and fourth phases are dependent upon cardiac weakness or defectiveness. They have made a proportion to indicate the numerical value of these two factors in any given case. These percentage values are calculated to a normal average pulse pressure of 45 mm. in which they found that the phases formed the following percentages of this pulse pressure:

First phase.....	33.1 per cent.
Second phase.....	44.4 per cent.
Third phase.....	11.1 per cent.
Fourth phase.....	13.3 per cent.
Thus 44.4 + 11.1 = 55.5 per cent., and	
33.1 + 13.3 = 44.4 per cent.	

Therefore, cardiac strength (C.S.): cardiac weakness (C.W.): 55.5:44.4. While this view is not yet accepted as consistently proven, nor generally employed as a basis for clinical investigation, nevertheless Swan³ has in his comprehensive study of 200 cases, done much to prove the

¹ *Loc. cit.*

² *Loc. cit.*

³ *Loc. cit.*

contention of the originators. Unfortunately, space will not permit of an extended review of this work. From it, however, we can, as suggested by Swan, draw the following conclusions:

"1. The auscultatory method is the most accurate and most convenient method of making blood-pressure determination.

"2. The first point should be considered the systolic or maximal pressure.

"3. The fifth point should be considered the diastolic or minimal pressure, except in cases in which a sound can be heard in the brachial artery, with no pressure in the cuff, in which case the fourth point should be considered to represent the diastolic pressure.

"4. The pulse pressure and the mean pressure can always be obtained by the auscultatory method.

"5. The systolic pressure obtained by the auscultatory method is almost always higher than that obtained by the palpatory method, and lower than that obtained by the graphic method.

"6. Whenever it is possible to work out the C.S. = C.W. ratio valuable information can be obtained concerning the functional ability of the myocardium at the time the observation is made.

"7. It may be said, as a rule, that a second phase forming more than 40 per cent. of the pulse pressure, and a C.S. = C.W. ratio, in which the C.S. factor is greater than the C.W. factor, give a fairly reliable index of the competence of the myocardium.

"8. Although one cannot make the C.S. = C.W. equation when one point or more is absent, a second phase of 40 per cent. or more of the pulse pressure indicates a good heart muscle and one capable of being put in better physiological condition.

"9. Absent points and tonal arrhythmias indicate cardiac muscle weakness."

Comparison of Results Obtained by the Auscultatory Method with Those Obtained by the Other Methods.—
Palpatory Method.—In practically every case the auscultatory method gives readings higher than by the palpatory. Korotokoff¹ found that the systolic readings by the former averaged from 10 to 12 mm. Hg. higher; Gittings² in a study of sixty-three cases found that in sixty-one the average systolic pressure was $16\frac{7}{10}$ mm. Hg. higher;

¹ *Loc. cit.*

² *Loc. cit.*

Warfield¹ obtained values of from 6 to 14 mm. Hg. higher. The greatest difference was noted in arteriosclerosis. Swan² studied the difference between these two methods in 100 consecutive cases, in ninety-eight of which the systolic pressure by auscultation was from 1 to 30 mm. Hg. below

Graphic	Auscultatory	Palpatory	
compared to	compared to	compared to	Graphic
160 + 21	139 + 13	126	-34
120 - 3	129 + 18	111	-15
120 + 8	112 + 2	110	-10
200 + 45	155 + 2	157	-43
128 + 31	97 + 5	92	-36
198 + 46	152 + 7	145	-53
182 + 28	154 + 14	140	-42
140 + 18	122 + 4	118	-32
158 + 23	135 + 10	125	-33
141 + 43	105 + 1	104	-49
164 + 47	137 + 7	130	-54
161 + 40	128 + 37	91	-97
150 + 55	95 + 0	95	-55
130 + 5	125 + 13	112	-18
138 + 28	110 + 5	105	-33
162 + 27	135 + 13	122	-40
178 + 26	152 + 7	145	-33

FIG. 26.—Comparative table of systolic readings made by the graphic method, the auscultatory and the palpatory methods, showing differences in millimeters between each method. Uskoff apparatus 12 cm. cuff (modified from Swan).

that given by the auscultatory method. The average difference was 9.98 mm. Hg. My own experience is in accord with these investigators.

Visual Methods.—Writers have found such wide difference in their own observations, due to the uncontrollable

¹ *Interstate Med. Jour.*, xix, 10, October, 1912, p. 860.

² *Loc. cit.*

elements of error, resulting from the type of instrument used, manner of application of the cuff and the personal equation of the examiner, that little if any dependence should be put in this method, either for systolic or diastolic determinations.

Graphic Methods.—Apart from the intricacy of these methods which renders them practically useless for clinical and bedside work, they have also been found to give misleading data, because sphygmographic tracings always give readings that are too high.

The Uskoff instrument for example gives a series of pulsations long before the blood-stream enters the artery below the cuff.

Swan has examined comparatively seventeen cases, by the graphic, the auscultatory and the palpatory methods, his results are shown in the accompanying table, Fig. 26, which has been modified by adding figures which show at a glance the variation between the methods in each case.

Additional Auscultatory Data.—The actual volume of the tones heard by auscultation depends on so many factors that their bearing and clinical significance can be practically estimated only by those experienced in this method of observation. This accomplishment when once mastered will be a never failing source of satisfaction and assistance to the clinician.

In auscultatory blood-pressure work, one should endeavor to follow and analyze the sounds in the same manner as when examining the heart. The examiner should take into consideration: *first*, the size and strength of the heart; *second*, the size of and the state of contraction of the arterial wall; *third*, arterial elasticity; *fourth*, the conducting

value of the stethoscope, the degree of pressure exerted over the artery and the thickness and tone of the overlying structures. A general knowledge of these elements will enable the careful clinician to arrive at finer points of decision in diagnosis, than if the auscultatory tone phases alone are depended upon.

Another advantage of the auscultatory method, beside its great accuracy, is the fact that the method is available in every case, regardless of the size of the artery, of the arm or the volume of the pulse. Further, it can be performed with any variety of sphygmomanometer and any stethoscope (small receiver preferred).

It should be borne in mind, however, that cases will be met in which it will be impossible to differentiate all the points and phases. The most usual anomaly according to Swan¹ is the *absent fourth point*. This is when, after the murmur has disappeared, the clear tap gradually diminishes until all sounds disappear. Sometimes the murmur is the first sound heard as the pressure diminishes in the cuff, *absent first point*. Sometimes the first point is heard but no murmur can be detected, suddenly the tap becomes dulled, and finally the sound disappears, *absent second and third points*. Again the murmur may be the first sound heard, and often it disappears, the tap diminishes in intensity, until the sounds disappear, *absent first and fourth points*. Sometimes all the points are absent except the *first and fifth, no phases*.

B. PALPATORY METHOD

Systolic Pressure.—After the pressure in the apparatus has been raised until the pulse is no longer palpable at the

¹ *Loc. cit.*

wrist, it is then gradually reduced; at the same time the level of the mercury or the position of the pointer is noted where the first pulse beat is felt at the wrist. This is the systolic pressure.

Diastolic Pressure.—As the air pressure continues to fall from a point below the systolic, the character of the pulse at the wrist is carefully noted. At first this is very feeble and thready in character and continues so for a time, when suddenly it assumes a full bounding character. At this moment the height of the mercury column, or the hand of the indicator, will represent the diastolic pressure in millimeters of mercury.

C. VISUAL METHOD

Systolic Pressure.—After obliteration of the pulse by the constricting cuff, it will be noted as the pressure is gradually released, that an oscillatory motion will be imparted by the pulse to the mercury column or of the needle of the indicator. The beginning of this oscillation has been believed to indicate the systolic point, as it was believed to indicate the approximate moment when the blood begins to pass beyond the constriction. With the advent of the auscultatory method it is now believed that readings taken by the visual method are only approximate and often inaccurate, as in many cases this movement appears many millimeters above the actual systolic point (see page 100).

Diastolic Pressure.—As the pressure is gradually released, the rhythmic motion of the mercury or of the indicator which appeared at the systolic point will be seen to gradually increase in amplitude up to a certain point, after which it decreases and finally ceases before zero pressure is

reached. During this phenomenon the diastolic pressure reading is taken at the maximum oscillation.

D. THE GRAPHIC METHOD

Systolic and Diastolic.—The determination of blood-pressure by the graphic method is accomplished by an apparatus which combines some form of mercury sphygmomanometer with a sphygmograph, whereby the movements of the mercury column and the level of the mercury are recorded upon smoked paper, or other suitable material, upon a moving surface or a revolving drum. This method, while thoroughly scientific, is not suited to clinical use (see page 84 for description) and will not be enlarged upon here.

E. THE OSCILLATORY METHOD

This method requires an instrument by which the changing character of the arterial impulse, as it passes under the cuff of the sphygmomanometer, is accentuated, so that variations in its force, transmitted to the apparatus by air currents, are transformed into visible motion in the instrument, when they are more easily analyzed and more accurately read. This method does not differ materially from the usual visual method, although it is said to be more accurate, and of particular value in studying low systolic and small pulse pressures.

Among those most commonly used instruments are those of Pal (sphygmoscope) and of Bing, both of which were introduced and described in 1906, also the instrument of Pachon, which is a modification of the aneroid apparatus, in which two series of compression chambers, one group large and one small, are used. The small one indicates the

pressure level and the large one actuates the oscillatory needle.

Fedde, several years later, introduced what he called a "Diastolic Indicator." This is simply a pith-ball oscillometer, in which the motion of the air, transmitted from the cuff to the indicator, is accentuated by means of a closed metallic chamber between which and the arm-band is interposed a vertical glass tube containing a delicate pith-ball, the motion of which visualizes the impulses.

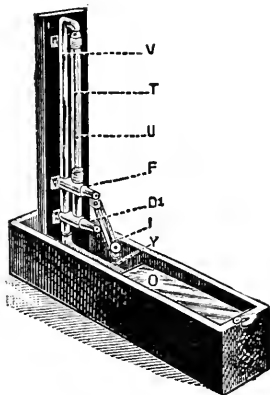


FIG. 27.—Fedde indicator as separate unit.

This instrument is shown in Fig. 27, where it is made as a separate unit for attachment to any variety of sphygmomanometer. The manufacturers of this instrument (G. P. Pilling & Son Co.) have also modified the construction of this instrument so that it may be included as a component part (at additional cost) in the Standard mercury sphygmomanometer.

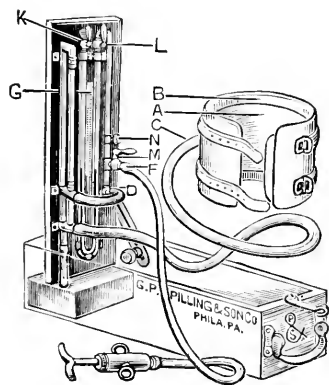


FIG. 28.—Sketch of standard mercury sphygmomanometer combined with Fedde indicator.

Reference to Fig. 28, will show the method of uniting the Fedde indicator to a sphygmomanometer. It will be noted that the narrow perpendicular glass tube contains a small light ball of pith or cork which is free to move up and down within the tube.

When determining the systolic pressure, pay no attention to this indicator, as each impact of air will make the ball dance violently but has no bearing on the test. When the pressure has reached the systolic point gradually release the air pressure when the ball will begin to move slightly in rhythm with the pulse. This motion gradually increases, until it reaches a maximum when quite suddenly its motion becomes markedly less. At the moment of this reduced movement the level of the mercury will indicate the diastolic pressure.

Diastolic Index.—In connection with blood-pressure tests it is interesting to consider a point brought out by MacWilliams and Melvin¹ and which may later develop into an additional diagnostic aid. These observers found it desirable to study what they have termed "the diastolic index." This is determined after the systolic reading has been taken, by making the diastolic reading both when the compression is being increased and decreased. They found a difference amounting at times to as much as 20 mm. between the diastolic readings obtained by these two methods. They believe that this difference gives important information concerning the liability of the pressure to vary in an abnormal way when a portion of the arterial tree is shut off from the general circulation. Similar changes have been noted by other observers in the systolic pressure when a large portion of the arterial system is suddenly obstructed (see Momberg constriction, page 225). The pulse rate also at times varies.

The demonstration of such marked changes would appear to suggest a poor response on the part of the vasomotor

¹ J. C. MacWilliams and G. S. Melvin, *Brit. Med. Jour.*, Nov. 7, 1914.

mechanism to accommodate by rapid changes in the caliber of the vessels for sudden changes in the volume of the circulating blood. This discovery has opened up a field for additional research, the value of the findings of which cannot be determined without further study and investigation.

The Subjective Method of Determining Blood-pressure.

—It has long been known that the subjective sensation of the patient may at times furnish a reliable guide in determining systolic and diastolic pressures. Thus when the cuff has been sufficiently inflated to obstruct the pulse, there is felt a slight impulse in the upper margin in the cuff corresponding to the line of the artery. As the pressure is lowered in the air system, thus pulsation proceeds downward so that it finally is felt below the cuff, where it measures the systolic pressure. Continued reduction in air pressure eventually results in disappearance of any sensation under or below the cuff. This indicates the diastolic pressure. While such readings may be quite accurate, this procedure involves a considerable and often an uncontrollable personal equation and should therefore not be depended upon.



Fig. 29.—Bracelet stethoscope for auscultatory blood-pressure reading.

The **Sphygmometroscope** will be found of great assistance in performing the auscultatory method. This instrument as shown in the accompanying cut (Fig. 29) is a Bowles stethoscope with a button-like projection attached to the face of the diaphragm. This greatly facilitates its application to the artery below the sphygmomanometer cuff,

where it is secured in position by a narrow metallic spring band similar to a phone operator's head-band. It is thus self-retaining and allows the operator the freedom of both hands with which to manage the sphygmomanometer.

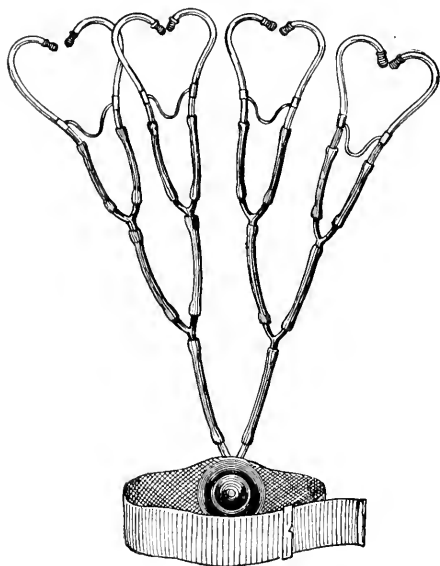


FIG. 30.—Multiple sphygmometroscope. Adaptation of multiple by stethoscope for auscultatory reading of blood-pressure.

This is quite important, as it will be found expedient to note the first disappearance of the pulse by palpation of the radial, as in the other methods, thereby preventing accidental or careless over-compression of the arm.

Fig. 30 represents a new device, first suggested by James F. Prendergast¹ which facilitates the teaching of blood-pressure readings by the auscultatory

method. The multiple binaurals permit anyone familiar with the sounds heard during auscultatory blood-pressure observations to direct the attention of a small group of observers during the actual performance of the test. No difficulty in hearing the sounds has been experienced from the greater distribution of the sound through a larger tubular system.

¹ J. F. Prendergast, *N. Y. Med. Jour.*, Vol. xevii, No. 2, 1913.

CHAPTER VI

THE NORMAL BLOOD-PRESSURE; ITS VARIATIONS AND THE PHYSIOLOGIC OR NON-PATHOLOGIC FACTORS AFFECTING IT

General Considerations.—In any attempt to discuss blood-pressure be it the systolic, the diastolic or the pulse pressure, viewed either separately or combined, we are at once confronted with the question, what constitutes a normal reading, what are its variations, and to what are they attributable? Also, how is the normal reading related to the clinical picture and when does it transgress the recognized limits of normal and become pathologic?

To intelligently and rationally apply the readings obtained in blood-pressure study, these questions must be thoroughly considered.

The clinical value of blood-pressure studies is in direct proportion to the experience of the attending physician, for only those who regularly make blood-pressure observations and record the results can hope to realize the full benefit to be derived from noting those smaller variations, which are often within normal limits, and whose value the casual worker will fail to appreciate even if he should detect them.

The veriest tyro will experience little difficulty in estimating the significance of great variations in pressure, but in such cases the observations are merely corroborative, as

the general clinical picture will usually furnish the same information equally well.

When it comes to a consideration of the variations in systolic, diastolic and pulse pressures, and their ever-changing relations, the problem becomes more complex, and not infrequently borders on a field that has been but slightly explored, and in which there is much opportunity for further original study and research.

At the outset it must be fixed firmly in the mind of the reader that the phenomenon of blood-pressure and its manifold variations is most complex. The more important facts relating to this have been considered in the chapter on physiology. We find it obvious from this study that there is no fixed normal pressure to serve as a standard, but that ever-varying conditions, both internal and external to the body, are continually at work, and that these result in a constantly varying blood-pressure picture.

Instrumental and Mechanical Variations.—Mechanical differences in instruments, apart from accidental error, due to defective manometers (which now are rare), must be considered, especially when comparison is made between figures obtained some years ago and now. In the early days of sphygmomanometry, the width of the arm-band, and the method of application of pressure were not critically considered, so that, except when indicated, it cannot be determined whether figures refer to pressure tests made with a 4-, 8-, 12-, or 16-cm. cuff, or whether any cuff at all was used, as with the early instruments of v. Basch and Potain.

All instruments employing the mercury scale or its equivalent (aneroids graduated to the mercury column in

mm. Hg.) will under the ordinary conditions give similar readings. The chief cause for inaccuracy is the use of cuffs of varying width. The standard cuff as accepted by most authorities is one having a width of compression surface of $4\frac{1}{2}$ to 5 in. (11 to 13 cm.) This, in all but the very obese will give uniform pressure readings, which by actual experiment have been found to correspond closely to the figures obtained by the direct introduction into a vessel of a canula communicating with a mercury manometer.

The cuff of Riva-Rocci as employed by Cook, in his simplification of the Riva-Rocci apparatus, measures 8 cm. in width. This has been found to interpose some resistance of its own, due to stretching of the rubber of the cuff, so that readings obtained by it are from 6 to 10 mm. higher (depending on the circumference of the arm) than those obtained by the standard cuff. Therefore, all figures obtained by the narrow cuff must be corrected, by the subtraction of 6, 8 or 10 mm. before they can be compared to the standard reading.

This difference has been carefully figured out by Chamberlain¹ and others and is only mentioned in passing as at this late date none but the standard cuff of 12 mm. should be employed.

The sphygmomanometer of Potain is not graduated in millimeters of Hg. at all, and therefore cannot be directly compared to the figures obtained by other instruments. Potain in his work on blood-pressure gives the normal with his instrument as 150 to 190 for men and 140 to 180 for women. The readings with the Gaertner tonometer range from 10 to 20 mm. below the standard.

¹ *Philippine Jour. of Sci.*, December, 1911.

THE NORMAL BLOOD-PRESSURE

The blood-pressure observation as it is made to-day includes an estimation of both the systolic and diastolic pressures from which the pulse pressure is computed, and any report of blood-pressure made within recent years should contain these figures, obtained by preference by the auscultatory method. This is fundamental, because, we know now that observations stating only the systolic pressure may be of relatively small value, since they often fail to convey any information as to the actual condition of the circulation; indeed they may even lead directly to an error in judgment, since it is not uncommon to meet cases in which the systolic pressure appears to be normal, yet if the diastolic and pulse pressures had been ascertained, a radical circulatory fault would have been discovered. On the other hand, an apparently abnormal systolic pressure, when accompanied by the diastolic observation, may show to a surprising degree a circulatory efficiency sustained by relatively normal pulse pressure.

It has already been stated that blood-pressure readings do not represent absolute values, for we know that all observations must, to a certain degree at least, be discounted, or better, be interpreted and adjusted to the whole clinical picture.

For convenience and for clinical study, certain rules have been formulated. These are based upon comparative clinical and experimental study, so that within certain limits it is now possible to state with fair accuracy the normal average pressure readings for any given age, together with their normal variations. This applies alike to systolic, diastolic and pulse pressure. We may also say in general

that, under normal conditions, the diastolic and pulse pressures, bear a certain arithmetical ratio to the systolic pressure, though even this is variable and considerable fluctuation is compatible with health.

Ignoring controllable sources of error, such as defective apparatus and the personal equation, and adhering strictly to observations made preferably by auscultation with the 12-cm. arm-band, we may down the following rules for guidance in estimating the degree of abnormality in blood-pressure.

The use of such guides or rules appears essential to the practical consideration of clinical cases, for without them the student or practitioner, who is unfamiliar with the employment of these tests, will have great difficulty in interpreting pressure values.

THE NORMAL VARIATIONS IN SYSTOLIC, DIASTOLIC AND PULSE PRESSURES

Age.—One cause of alteration in blood-pressure, which is more or less constant and progressive in nature, is that incident to increasing years, and is shown by a gradual elevation in the average systolic and diastolic blood-pressure in which the diastolic participates somewhat less than does the systolic. The result of this unequal change is a gradual increase in pulse pressure.

While innumerable statistics are available they are not all equally reliable. Among the best is the work of Woley,¹ which summarizes a clinical study of a thousand normal persons between the ages of fifteen and sixty years.

The results are shown in the chart (see Fig. 3) which

¹ *Jour. A. M. A.*, Vol. lv, 2, 1910, p. 121.

gives also the general average systolic pressure for any age within these limits, also the average high and low pressures, together with the average for males and for females at all ages. Except for slight and inconsequent differences, these figures are in close accord with those of other recent investigators.

There are a few points worthy of notice that might be mentioned here, namely, in regard to a rapid or slow pulse.¹ The average blood-pressure in all cases with a pulse under 65 was 123 mm., while the average blood-pressure in all cases with the pulse over 85 was 130 mm. It was also noticed that there were twice as many healthy individuals with a pulse over 85 as there were with a pulse under 65.

In my recent teaching on this subject, it has been my habit to state that the average systolic pressure for males of twenty years is 120 mm. Hg., and to suggest the employment of a formula devised to give approximately the average systolic reading for any age, as affected by that factor.²

The only change since made has been that of phraseology which now eliminates fractions. In these six years I have found no reason to alter the formula which has been adopted in many many directions including some of our larger insurance companies. The formula is as follows: "Consider the normal average systolic pressure at age twenty to be 120 mm. Hg., then add 1 mm. Hg. for each additional two years of life over age twenty." Of course it is realized that figures obtained by this or any other arbitrary formula must be endowed with great elasticity, and must at times

¹ Woley, *loc. cit.*

² "The Sphygmomanometer and Its Practical Application," 1st edition, 1909, The G. P. Pilling & Son Co., Philadelphia.

be so modified by so-called physiologic factors as to lose entirely this relation.

The Systolic Pressure in Childhood.—There is a certain definite relation between the age of the child and the height of pressure. The younger the child the lower the pressure, yet the tallness of the child often has a greater bearing on the systolic pressure than has the body weight. Sex plays but an insignificant part, as we but seldom meet differences of more than 5 mm. attributable to the sex factor.

The blood-pressure in children will be found to vary under the same conditions and influences that operate in the adult except that the physical development factor is often more powerful in proportion. The actual difference in pressure readings made at different times will not be as great as in adults on account of the relatively lower systolic pressure.

The diastolic pressure is usually between 25 and 35 mm. below the systolic, which means that the pulse pressure is between 25 and 35 mm.

My own observations lead me to believe that puberty in a normal well-nourished child, is the beginning of the established adult pressures as found in the adult. I have often met pressures of S. 110–D. 80– PP. 30 in children of from fifteen to seventeen years of age. The following composite table will give a fair indication of the average systolic pressures between the ages of one and seventeen years.

Systolic pressures in millimeters of mercury (9-cm. or 12-cm. arm-band, depending on size of arm).

Age	Mm. Hg.	Age	Mm. Hg.
Up to 1 year.....	70–85	Up to 10 years.....	90–105
Up to 2 years.....	75–90	Up to 13 years.....	95–110
Up to 4 years.....	80–95	Up to 15 years.....	95–115
Up to 6 years.....	80–100	Up to 17 years.....	95–120

The averages as obtained in my own observations, while not in absolute accord with this table or with other individual reports, are no more at variance than those of other reporters.

It will be found extremely difficult to make accurate records in children under three years of age, even of the systolic pressure alone, while to determine the diastolic is at times impossible.

In children except in extreme conditions the diastolic pressure is of little importance, as this observation attains its greatest value in chronic and constitutional diseases which are rarely met in early life.

Diastolic Pressure in Adults.—Until recently the study of the diastolic pressure received but a fraction of the attention which it deserves, because of the unreliability of the readings, this being the result of the great variety of methods heretofore employed in blood-pressure studies and also because of the scarcity of reports of sufficiently large series of diastolic observations from which to obtain reliable averages. From the study of a large number of observations which I have recorded, in which the auscultatory method alone was used, I have reached the conclusion that a working formula may be safely employed, if not applied too rigidly, which will give an approximate indication of the normal diastolic pressure in relation to any given systolic pressure in health, and that it can be used as a guide to separate the normal from the pathologic blood-pressure values. Briefly stated this formula is as follows: "The diastolic pressure will appropriate in value two-thirds of the systolic pressure and the pulse pressure will be one-third of the systolic pressure or one-half of the diastolic pressure."

This gives a systolic, diastolic and pulse pressure ratio of 3:2:1.

Again let me reiterate that these values are not absolute and should not be so considered. They must often be

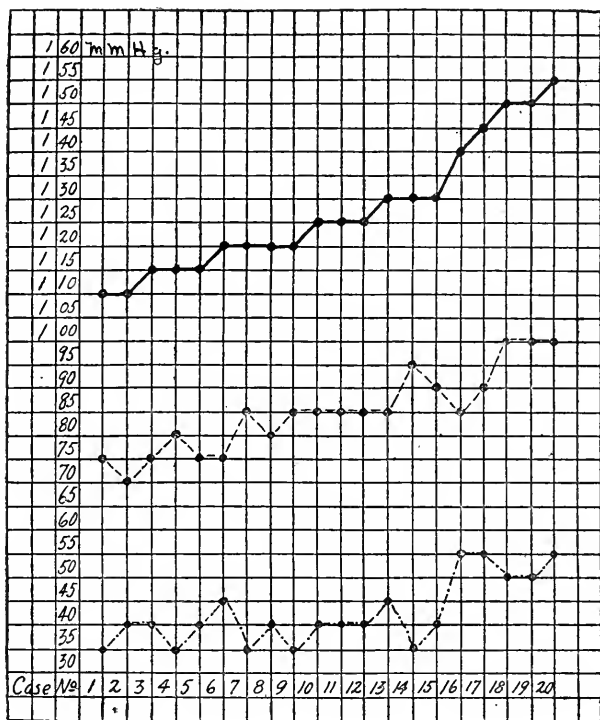


FIG. 31.—Shows a series of blood pressure readings in twenty normal individuals selected at random. The individuals range in age from 22 to 53 years. A mathematical average of the twenty readings gives an average systolic pressure of 125 mm. Hg., a diastolic pressure of 85 mm. Hg., and a pulse pressure of 43 mm. Hg.

much modified, to meet those many non-pathological influences to which the circulation is constantly subjected, and which will be discussed in detail below.

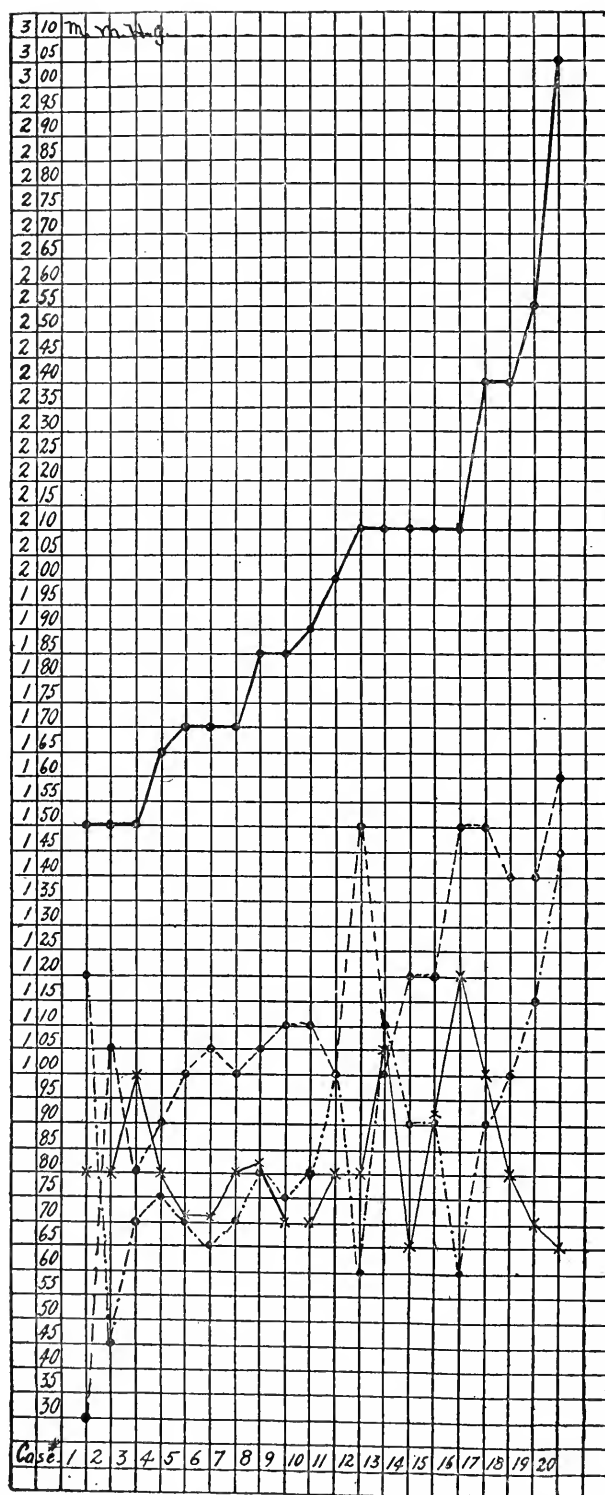


Fig. 32.—For explanatory note, see following page.

For illustrative purposes, in a paper appearing in the *New York Medical Journal* (Feb. 27, 1915) I selected at random twenty complete observations made upon twenty normal individuals between the ages of twenty-two and fifty-three. These cases were all in good health, but in this series no particular effort was made to exclude the usual normal causes of variation (see Fig. 31). By consulting this table, we find the highest systolic pressure was 155, the lowest 110. The lowest diastolic pressure 70, and the highest 100. The greatest pulse pressure was 55 and the smallest 35. The average figures for the twenty cases were systolic pressure 125, diastolic pressure 85, pulse pressure 43 (excluding fractions) which conforms almost exactly to the 3:2:1 ratio (for comparison with abnormal series, see chart (Fig. 32)).

Sex.—The average systolic pressure for women in a series of 2000 observations recorded by Theodore C. Jane-way, was 10 mm. less than that of males. In children this variation rarely exceeds 5 mm. The diastolic pres-

FIG. 32.¹—Represents a series of blood-pressure readings in twenty abnormal individuals. In each case the initial observation was used, in order that it might not be influenced by therapeutic measures, but would represent the state of the circulation resulting from the pathological condition. Much interesting information may be derived from a comparison of the two charts, the more important points of which are discussed below.

1, I. C., age 23, m., double aortic. 2, E. F., age 71, f., mitral regurgitation. 3, C. H. W., age 62, f., angina pectoris? 4, Edwards, age 74, f., chronic myocarditis. 5, J. A., age 58, f., myocarditis, chronic. 6, R. McC., age 50, f., pyorrhœa, autointox. 7, W. M., age 69, m., Chr. interstitial N. 8, A. J., age 63, m., arteriosclerosis. 9, B. M. McG., age 64, m., Chr. myocarditis. 10, Dr. M., age 69, m., arteriosclerosis. 11, W. B., age 65, m., arteriosclerosis. 12, E. W. C., age 54, m., cardiovascular. 13, J. F., age 55, f., chronic myocarditis. 14, R., age 60, f., myocarditis, chronic. 15, E. S. T., age 67, m., arteriosclerosis. 16, M. J. S., age 36, m., acute nephritis. 17, F. M. R., age 46, m., chronic interstitial. 18, S. F. R., age 42, m., arteriosclerosis. 19, C. B. Y., age 60, m., cardiovascular. 20, S., age 52, m., arteriosclerosis.

¹Solid black line represents systolic pressure, broken black line represents diastolic pressure, line of dots and dashes represents the pulse pressure, and solid line with crosses represents pulse rate.

sure is relatively higher in women for the same age, due chiefly to their generally poorer muscular development; as a result, the pulse pressure averages about 5 mm. less in women than in men.

Size, Muscular Development and Body Strength.—The physical fitness and muscular development of the individual affects slightly the average normal blood-pressure readings. It has already been stated that, because of generally poorer muscular development, the systolic pressure in women is lower than in men. A difference may

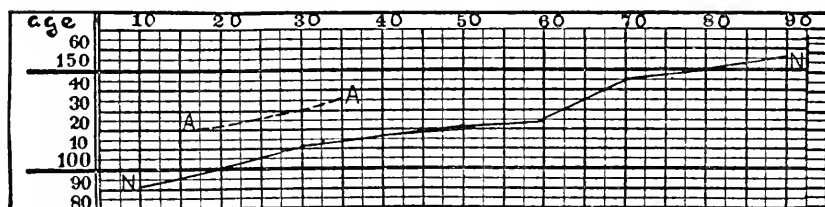


FIG. 33.—Chart showing averages of blood-pressure at different age periods. The line A-A indicates a higher than average blood-pressure in trained athletes (Barach and Marks).

also be noted between the average pressure in men of poor, as compared with those of robust, physique; as for example, between the sedentary clerk and the trained athlete. A difference of from 10 to 15 mm. in the systolic pressure and a proportionate variation in the diastolic and pulse pressure may thus be accounted for.

A recent study by Barach and Marks¹ throws some light upon the subject of absolute strength in comparison with the average systolic pressure. This work was per-

¹ Joseph H. Barach and W. L. Marks, *Arch. Int. Med.*, May, 1913, No. 2, p. 485.

formed on forty-eight students between the ages of fifteen and thirty, with the following results:

Average Systolic Pressure as Compared with Strength.

—Total strength (intercollegiate standard) range between 704 and 541 kilo. Average systolic standing was 124.5 mm. Average diastolic 79.35. Pulse pressure 44.91. Total strength between 505.4 and 355.9 kilo, average systolic pressure 120.57, average diastolic 74.61, average pulse pressure 45.96 (see Fig. 33).

There is seldom any appreciable variation between the pressure readings in the two arms, except in cases too rare to be considered, neither does the muscular development of the arm or the size of the part have any influence on the blood-pressure reading, although an extremely large arm with a too narrow band may give readings which, due to these elements of error, are too high. The same may be said of pressure readings, made upon a part that is the seat of edema.

Temperature, Emotion and Excitement.—The statement of certain observers to the contrary, it seems very unreasonable and hardly likely that the temperamental characteristics of an individual can produce any permanent variation in the average pressure level; for example we detect no appreciable difference in the average readings in the nervous as compared with the phlegmatic type.

There is no doubt, however, regarding the marked influence of temperament upon the degree of response to external stimuli, both physical and psychic; thus the nervous high-strung individual will show a greater response to excitement as shown by a transitory rise in blood-pressure than will the placid individual. Judson

Daland,¹ cites a case of a person driving an automobile through a crowded thoroughfare with a resulting rise of 20 mm. in the systolic and 15 in the diastolic whereas the same individual driving through a quiet street experienced a rise of but 15 in the systolic.

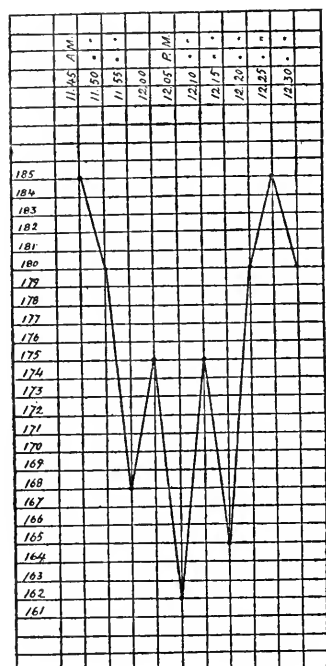


FIG. 34.—Rapid variation in systolic blood-pressure, occurring in forty-five minutes, patient sitting quietly in office.

The disturbing influence of nervous excitability is frequently met while endeavoring to ascertain the true pressure levels in nervous and high-strung persons. In interpreting pressure readings made when it is plainly impossible to eliminate this psychic influence, allowance must be made for discrepancies of this origin, particularly when such readings show abnormal variations within a limited space of time (see Fig. 34).

Severe pain may increase the systolic pressure from 15 to 25 mm. Mental excitement and anger may cause an even greater rise.

The effects of excitement, apprehension and fear are seen repeatedly in daily practice, where the initial reading may be surprisingly high, while later readings, as the patient becomes accustomed to the procedure, are found to maintain a lower level (see page 355).

¹ *Penna. Med. Jour.*, July, 1913.

In such cases, much may be done to eliminate these factors by establishing cordial relations between the patient and the operator, and by carefully explaining the technique of the observation and the amount of discomfort to be expected.

Posture.—O. Z. Stevens¹ reports a series of observations upon twenty healthy medical students, in which the systolic pressure and pulse rate alone were studied in all conceivable postures.

Ten Centimeter Cuff. Pressure in Millimeters of Mercury

*Systolic Pressure and Pulse Rate*²

Systolic pressure	Standing	Sitting	Supine	Head down	Right lateral	Left lateral
Right arm.....	132.6	133.3	152.5	166.2	155.0	110.0
Average.....	130.8	131.7	150.4	165.6	143.5	133.0
Left arm.....	130.0	130.0	148.3	165.0	141.0	156.0
Pulse rate.....	86.0	82.0	68.7	65.8	68.1	69.1

An extensive experimental study already referred to³ covers the effect of change in posture, without physical exertion, upon the pressure, from which article the accompanying table is taken.

Summary.—In changing from the erect to the horizontal, the systolic pressure will usually be increased. The diastolic pressure will almost always be diminished. When the erect posture is resumed, the systolic pressure will almost invariably fall and the diastolic rise.

¹*loc. cit.*

²O. Z. Stephens, *Jour. A. M. A.*, Oct. 1, 1904.

³Barach and Marks, *loc. cit.*

Effects of Changes of Posture on Arterial Pressure.—(*Barach and Marks*)*Subject at Rest on a Movable Table*

Maximal pressure			Than first.	Minimal pressure			Than first
Erect	Horizontal	Erect		Erect	Horizontal	Erect	
×	28+ 20—	7+ 40—	8+ 38—	×	4+ 44—	43+ 4—	25+ 21—

First column: × = height of maximum pressure in erect.

Second column: + = increase of maximum pressure when changed to horizontal.

Second column: — = fall of maximum pressure when changed to horizontal.

Third column: — = fall of maximum pressure when changed to erect.

Third column: + = rise of maximum pressure when changed to erect.

Fourth column: — = the height of maximum pressure was lower when the erect posture was resumed, than the first readings in the erect.

Fourth column: + = height of maximum pressure greater than first.

Pulse Pressures.—These range between 15 and 72, the average being 36.46, being greater when the maximum pressure is higher. Arranged according to age, in the erect posture it was 37.1 between ages sixteen and twenty-one, and 40.61 between ages twenty-one and thirty.

Effect of Change of Posture on Pulse Pressure.—From erect to horizontal an increase in 40 and a fall in 7. From horizontal to erect a decrease in 41 and increase in 4. Finally, it remained higher than the first reading in eleven cases. The pulse pressure seems to vary in the same direction as the maximum pressure.

When considering the effect of posture upon blood-pressure readings one should not confound this with the effect of gravity upon the fluid in the arterial tree, which is the same as might be expected in any problem in hydrostatics. This source of error may be eliminated by keep-

ing the arm in such a position that the cuff is always approximately at the heart-level, irrespective of the position of the patient, and also by making all subsequent observations on the same patient, in the same posture and under the same condition as far as practicable.

Sleep and Rest.—Prolonged rest in bed for one previously active, especially if there be a tendency to high pressure, will result in a rapid and sometimes in a marked fall in systolic pressure, with a production of a new and lower systolic level. Brooks and Carroll¹ do not believe that physical rest alone, either in normal or in high-pressure cases, is responsible for this effect upon the systolic pressure but rather that the result is due to the psychic and mental rest accompanying the physical, and that these factors are accountable for the undoubted benefit derived from absolute rest in high-pressure cases. While the effect of sleep is well known to cause a marked fall in systolic pressure (*vide infra*) these investigations have proved that the total average twenty-four hours systolic pressure shows little variation whether the patient be confined to bed or not. For this reason they do not believe that the so-called "sleep drop" can be used therapeutically in order to lower blood-pressure.

In the examination of normal individuals (twenty-nine with high and thirty with low blood-pressures) they found that the greatest sleep drop occurred during the early hours amounting to as much as 24 mm. and that three hours after an awakening there was still an average depression of 12 mm. The greatest fall occurred in those having the highest initial pressure.

¹ *Arch. Int. Med.*, August, 1914.

Disturbance of sleep during the first hours often delayed but did not necessarily prevent the maximum fall, although frequent interruptions were likely to prevent it.

The relation of diastolic and pulse pressure to sleep and rest has not been studied sufficiently to warrant conclusions, but owing to the general vasomotor relaxation which accompanies profound sleep it is probable that the diastolic pressure falls proportionately more (not in actual millimeters) so that the pulse pressure is relatively larger during the early hours of the sleep than in the daytime.

Diurnal Variations.—In close relation to the preceding, but not definitely a part of it, are the changes in pressure which occur during the waking hours, incident to ever-present and constantly varying physiologic factors.

Commencing with a depression of 10 or 12 mm., below the average systolic, found in the early morning hours, the pressure gradually rises and reaches a maximum usually between 3 and 7 P.M. This rise is held to be independent of the effect of food and of the digestive processes. Erlanger and Hooker¹ have found that the rise in systolic pressure is greater than the diastolic rise, resulting in a gradually increasing pulse pressure as the day advances. The range of maximum pressure varies considerably in different individuals, but according to Weyssse and Lutz² the highest and lowest maximum pressures are practically equidistant from the average pressure of any one individual.

The influence of the non-pathologic or physiologic factors

¹ *Johns Hopkins Hos. Rep.*, 1904, Vol. xii, No. 53.

² A. W. Weyssse and B. R. Lutz, *Am. Jour. Physiol.*, May, 1915, xxxvii, No. 2.

may so influence a day's series of observations that they may not follow the usual rule. An example of this is shown in Fig. 35. Physical and mental strain, the ingestion of food and fluids will produce striking and rather rapid alterations (see page 132). Janeway has reported¹ daily systolic variations amounting to 60 mm., although personally I have seen none to equal this.

Periodic Variations.—The daily pressure curve is subject to intrinsic variations of a more or less automatic character. Thus the respiratory movements may be demonstrated in graphic tracings. During quiet respirations these may be so slight as to pass unnoticed, although at times violent respiratory movements may interfere with the accurate determination of the systolic point by auscultation. The maximum variations which may be attributed to this factor rarely amount to more than between 5 and 10 mm.

It is found that the highest point of the respiratory curve occurs at a point midway between expiration and inspiration. The Traube-Herring waves, which have been attributed to chemical changes occurring in the blood, occur rhythmically at intervals of a few minutes.

¹ Theodore C. Janeway, "The Clinical Study of Blood-Pressure," 1904.

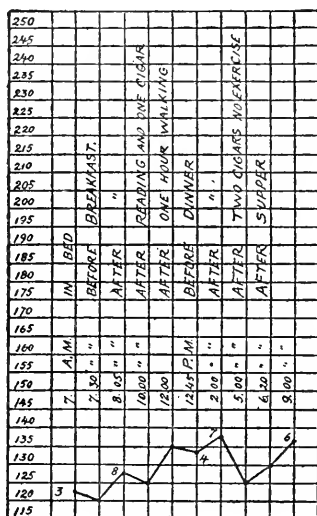


FIG. 35.—Record of systolic pressure variations occurring during the working hours of a young healthy man.

These variations are more of academic than of clinical interest.

Physical Exercise and Exertion.—Exercise results, in the normal individual, in an elevation in the systolic blood-pressure, a less proportionate rise in the diastolic pressure with a consequent increase in the pulse pressure and an acceleration in heart rate.¹

Exhaustive studies have been made to determine the amount of actual energy expended as measured by an ergostat, as compared with the measured changes in the blood-pressure, although so far nothing of clinical value has been published.

When exercise is moderate and prolonged, as in walking, the systolic pressure will rise from 5 to 15 mm. when it appears to become adjusted to a new level, upon which additional exertion of the same kind has little if any further effect, until a condition of extreme fatigue is reached. Fatigue from prolonged muscular effort results in a fall in systolic pressure which is not immediately arrested by stopping the exertion but which may progress until a dangerous reduction ensues.²

The degree of muscular exertion and the suddenness of the effort affects the rapidity and the degree of systolic rise. Sudden severe exertion has been shown to cause a systolic rise of 50 mm., while severe exertion causes an average rise of from 25 to 30 mm. It is also said that the degree of mental effort accompanying the physical exertion influences this rise, and that it is further increased by the mental effort. When exercise ceases (if

¹ Krehl "Clinical Pathology," 1905, 3d edition.

² Karrenstein, *Zeits. f. klin. Med.*, 1903, i, p. 322.

stopped short of fatigue) the pressure rapidly regains its normal level as does also the pulse rate.

Edward O. Otis, from a study of a large number of athletes, believes that a persistent fall in pressure following prolonged and violent exertion is evidence that the exertion has been too great or too prolonged. In this same connection O. S. Lowsley,¹ also studying healthy athletes in training, found that the systolic pressure and the pulse rate were usually increased during exertion and were found to be above the original figures even at the end of periods of prolonged and exhaustive work, provided the observations could be made before the subsequent fall set in. After the cessation of exercise there is usually a period of subnormal pressure, and the more exhausting the work and the more prolonged the effort the more marked and prolonged the subnormal phase.

There is often observed a secondary rise in pulse rate during the period of subnormal pressure, the significance of which has not yet been fully determined.

Very violent exercise even for a few seconds, as in a 100-yard dash, may cause a more prolonged and profound subnormal phase, than does more prolonged but more moderate work.

Lowsley suggests that the duration of the subnormal phase may be taken as a fair index of the strain on the circulatory system, and states that he has been led to believe that if the subnormal phase lasts less than an hour, it may be considered to be within the hygienic limit, but that if it persists for more than two hours it is a sign that the margin of safety has been exceeded.

¹ *Amer. Jour. Physiol.*, Mar. 1, 1911.

Passive Movements and Massage.—Passive movements, performed either by attendants or by special machinery devised for performing such work, cause no significant rise in blood-pressure; on the contrary, in high-pressure cases, they may be relied upon to reduce the pressure. The effect of such measures on diastolic and pulse pressure has not been sufficiently studied to base reliable conclusions upon.

Massage even when applied over the abdomen has not been shown to cause an appreciable rise in systolic pressure¹ (see also page 441).

Altitude and Atmospheric Pressure.—B. P. Pomeroy² states that nearly all clinical and experimental data show that a reduction of barometric pressure causes a lowering of both systolic and diastolic blood-pressure and that this effect is not transient but persists during the continuance of the low barometer. The reduction in pressure is usually accompanied by a slight rise in pulse rate. F. C. Smith,³ from studies of 250 normal individuals at an altitude of 6000 ft., as compared with the reports of studies by competent observers made at sea level, believes that the effect of altitude and barometric pressure is overestimated, and sometimes confused with that of other influences; nevertheless, the average fall reported by other observers taken after the sharp variation, due to sudden change have been compensated, while present rarely exceeds 10 mm. Bearing on the same subject, is H. Brooks'⁴ study of the blood-pressure of seventy-five

¹Eichberg, *Jour. A. M. A.*, Sept. 19, 1908.

²*Int. Med. Jour.*, Vol. xviii, 1911, p. 731.

³*Jour. A. M. A.*, May 29, 1915, Vol. lxiv, No. 22.

⁴*Med. Rec.*, May 25, 1907.

compressed-air workers before, during and after working in caissons. He found a rise in arterial pressure while working under compressed air (+31 lb.) but noted that the variation in systolic pressure afterward, was no greater than that found under usual atmospheric conditions, neither was the pulse pressure altered; therefore moderate degrees of heart and kidney disease are not contra-indications to working under compressed air.

The Taking of Food, Drink and Digestion.—Weyssse and Lutz¹ found that a rise in systolic pressure, averaging 8 mm. Hg. occurs immediately after the ingestion of food. This maximum elevation according to M. Loeper,² persists for from fifteen to forty-five minutes, after which it falls to a point slightly below the normal, while after the third hour a second elevation was regularly noted. (Weyssse and Lutz did not note this.) Loeper analyzes the different elements which may be responsible for these fluctuations. Thus the initial hypertension seems to be due exclusively to the distention of the stomach, and an increase in intra-abdominal pressure, as it is more marked the larger the amount eaten and drunk. The secondary hypertension corresponds to the active period of gastric secretion (splanchnic hyperemia) and was found more marked after foods that stimulate gastric secretions, being strongest with a meat-containing meal, weakest with milk and very transient with water and macaroni. The secondary hypertension corresponds to the distention of the intestines and is probably the result of the rapid absorption of digestive substances, having a directly hypertensive action upon the

¹ *Loc. cit.*

² *Arch. des Maladies du coeur*, March, 1913.

blood-vessel walls or a stimulating effect upon the chromaffin system (author's conclusion).

H. L. Higgins¹ attempts to explain the effect of food and digestion upon blood-pressure by the influence of these processes upon alveolar carbon dioxide and the effects of such changes on the respiratory and cardiac centers. He states that alveolar CO₂ rises on taking food and remains high during active digestion, and that a high alveolar air content is coincident with vasodilatation while a low alveolar CO₂ tension causes vasoconstriction.

These facts would seem to demonstrate the advisability of avoiding large meals or imbibing much fluid in diseased conditions accompanied by high pressure. For the same reason, stimulating foods are to be avoided especially by those with arteriosclerosis and chronic kidney disease.

Erlanger and Hooker have noted a constant increase in pulse pressure during the active digestive process.²

The ingestion of a large amount of fluid, particularly of beer, is usually followed by a transitory rise in systolic pressure, which may amount to from 10 to 20 mm.

Influence of Menstruation.—At the onset of this phenomenon there is a fall in systolic pressure reaching its lowest level (a reduction of from 15 to 20 mm.) at the height of the period; while the normal level is again attained three or four days after the cessation of the flow.

Effects of Changes in Intra-abdominal and Intrathoracic Pressures.—Owing to the close relation of the abdominal and thoracic cavities to the larger blood-containing areas, it seems advisable to include a study of changes of pressure

¹ *Am. Jour. Physiol.*, April, 1914, xxxiv, No. 1.

² *Johns Hopkins Hos. Rep.*, 1904, xii.

in these regions and their effect upon blood-pressure in this chapter (see also Chapter XX, page 356).

H. Emerson¹ states that there exists an intra-abdominal pressure in the normal human which is above atmospheric pressure, and that this fluctuates with the movements of the diaphragm, also that anything which increases the intra-abdominal pressure will cause a fall in general systolic pressure. This is apparently due to diminished flow of venous blood into the vena cava with a resultant diminution in the systolic output of the left ventricle.

J. A. Capps² reports that drainage of fluid from the thoracic or abdominal cavities is, as a rule, accompanied by a fall in systolic pressure, averaging 32 mm. and that the greatest depression usually comes several minutes after the needle is withdrawn, though it may occur at any time during the procedure. The amount of fall seems to bear a direct relation to the quantity of fluid removed and to the rapidity with which it is withdrawn.

Occasionally the fall is very slight. In one case following the removal of 3000 c.c. of fluid, a fall of 62 mm. was noted. The residual fall in systolic pressure observed one hour after the removal of the fluid averages 12 mm., which corresponds to the average fall of 10 to 14 mm. in intra-abdominal pressure after tapping.

A sudden and marked fall may result in temporary collapse, due probably to a sudden relaxation of the splanchnic area. Such an incident can be guarded against by repeated blood-pressure observations during tapping, and may often be prevented by external pressure upon the

¹ *Arch. Int. Med.*, June, 1911.

² *Jour. A. M. A.*, 1907, xlviii, No. 1.

abdominal wall which will cause an immediate rise of 5 to 20 mm. Hg. in systolic pressure.

Posture is important, as normally the pressure is higher in the sitting than in the recumbent posture, although at the end of paracentesis, the mere fact of lying down often increases the systolic pressure from 6 to 20 mm.

Effect of Vomiting on Blood-pressure.—According to the experimental studies of Brooks and Luckhardt¹ vomiting occasions marked changes in the circulatory system. There is seen sometimes a period of elevated pressure, but more frequently, a sudden and enormous drop in systolic pressure with cardiac inhibition, at the moment of emesis. There are always great oscillations in blood-pressure. These great and sudden variations, might cause a rupture of blood-vessels which would not occur with the same pressure but with slower changes.

¹ C. Brooks and A. B. Luckhardt, *Am. Jour. Physiol.*, January, 1915, xxxvi, No. 2.

CHAPTER VII

THE PULSE PRESSURE

While the systolic pressure has been receiving great attention from the general practitioner the other phenomena of blood-pressure, the diastolic pressure, and the pulse pressure, which is the difference between the systolic and diastolic pressure have, until quite recently, received but scant attention. Possibly this is due to the fact that the methods of measuring the diastolic pressure have not been uniform and thus no comparison was possible between the results of different observers, while even those of a single observer were so variable that no conclusions could be drawn.

It is now accepted that the auscultatory method of determining blood-pressure is the most accurate and uniform of all the methods available for bedside work, so the early difficulty no longer exists. There appear to be two schools of teaching (see page 90) regarding the method of determining the pulse pressure by auscultation, one of which teaches that the disappearance of all sound is the diastolic point, the other that the point of transition from the third to the fourth phase (fourth point of Swan) is the diastolic point. The difference between these two methods is sufficient, except in the exceptional case when the change from fourth to fifth point is almost simultaneous, to alter to an appreciable degree the pulse pressure.

This difference, according to Swan,¹ averages 14 mm., being, of course, lower when the fifth point is taken as the criterion. Accepting 45 mm. as a fair average pulse pressure it is evident that the difference in reading resulting from differing interpretation, can amount to $33\frac{1}{3}$ per cent. more of the whole. Throughout this work, unless otherwise specified, the data employed are based upon diastolic readings made at the fifth point, that is at the moment of disappearance of all sound.

Concerning the importance of a knowledge of the pulse pressure, it seems logical to believe that the pulse pressure, which is the actual head of pressure driving the blood into the peripheral vessels, and which represents the systolic output of the heart, should be of first importance in cardiac prognosis (see functional tests, Chapter XIX, page 331).

The Determination of the Pulse Pressure.—The pulse pressure cannot be determined until both the systolic and the diastolic pressure have been estimated, as it is computed by subtracting the diastolic pressure from the systolic pressure, and the accuracy with which these are computed will determine the accuracy of the pulse pressure. We believe the pulse pressure to be highly important because it is not so much the knowledge of the actual pressure (systolic) under which an organ receives its blood, as it is the knowledge of the amount of blood received by the organ per unit of time, that enables us to judge of functional capacity. The volume of blood delivered by the heart to the great vessels per minute, even if not actually measured will be indicated by the pulse pressure, times the heart rate per minute. At the same time this calculation

¹*Loc. cit.*

indicates the velocity of the blood, for we believe that, within certain fairly restricted limits, there is a relation between pulse rate, pulse pressure and blood velocity, the accuracy of this opinion has been demonstrated, experimentally as well as theoretically.¹

It has also been shown that a reduction in pulse pressure means a lessened velocity of blood flow, although the reverse is not necessarily true.

The Relation of Pulse Pressure to Other Pressure Values.—Clinical evidence points clearly to the fact that in the normal adult, the pulse pressure approximates one-third of the systolic pressure and one-half of the diastolic pressure, thereby giving a relation of 3:2:1 for the systolic, the diastolic and the pulse pressure (see chart, page 117), although slight variations from this are compatible with a normal cardiovascular system. Upon the other hand, practically all those conditions which tend to an elevated systolic pressure of 180 or more disturb this ratio, as do also conditions of low pressure, accompanied by vasomotor weakness, so that in many chronic cardiovascular and renal conditions and in shock, the pulse pressure tends to approximate the diastolic pressure and may at times equal or even exceed it (see chart, page 118). This statement should not be held invariable, as Warfield² has cited cases of myocardial disease with normal blood-pressure values five minutes before death.

The normal average pulse pressure may be considered to lie between 35 and 50 mm.

Low Pulse Pressure.—The most consistent low pulse

¹Louis M. Warfield, *Jour. A. M. A.*, Oct. 4, 1913, lxi, 14, p. 1254.

²Louis M. Warfield, *Amer. Jour. Med. Sci.*, December, 1914, cxlviii, 6.

pressures are met in cases of pulmonary tuberculosis, in typhoid fever between the third and fifth weeks, and in many wasting diseases and exhausting fevers. The reduction of pulse pressure in these cases is accomplished chiefly at the expense of the systolic pressure, which is reduced relatively more than is the diastolic pressure.

Large Pulse Pressure.—According to W. J. Stone¹ the pulse pressure is usually increased in anemia. As examples he cites three cases in which the systolic blood-pressure varied from 120 to 135 and the diastolic pressure from 60 to 85, giving the pulse pressure 45 to 65. The patient with the lowest red count (1,630,000) had the lowest diastolic pressure and in consequence a pulse pressure of 65. The pulse pressure in aortic insufficiency is always large, probably larger than in any other condition (see chart, page 332).

In chronic nephritis and in arteriosclerosis with good cardiac compensation the pulse pressure is increased, while in myocardial insufficiency it varies depending apparently upon the size of the heart, being high where the heart is hypertrophied and small where the heart is small. As a general rule it may be stated that in general blood-pressure depression, irrespective of whether the preceding pressures were above normal or not, the presence of a small pulse pressure usually means a gravely affected heart and an unfavorable prognosis.

Significance of Pulse Pressure Changes.—In general, when the systolic pressure is normal, an excessive pulse pressure signifies peripheral dilatation. When the systolic pressure is high, the increased pulse pressure is to be

¹ *Jour. A. M. A.*, Oct. 4, 1913, lxi, 14, p. 1256.

regarded as normal and of no special significance, especially if the 3:2:1 ratio is approximately maintained. Decreased pulse pressure indicates marked peripheral constriction and may be an unfavorable prognostic sign; as in disease conditions a decreasing pulse pressure usually means cardiac failure. This condition often occurs in the latter stages of the high-systolic-increased-pulse pressure cases, in which the occurrence of a low-systolic pressure and an increased pulse pressure usually means circulatory failure through loss of vasomotor tone and heart-muscle weakness. This is seen in febrile conditions, *cachectic* states, malignant disease and in convalescence from long illness.

The Blood-pressure Quotient.—It is believed that the pulse pressure, divided by the systolic pressure, gives an indication of the parts played by vascular resistance and heart work respectively (see Functional Tests, page 331). The determination of the blood-pressure quotient is based upon the belief that increased heart work is shown by an increase in systolic pressure accompanied by an enlarging pulse pressure, while on the other hand an increase in peripheral resistance alone will increase systolic pressure and decrease pulse pressure. In addition reduced peripheral resistance lowers systolic pressure and increases pulse pressure; therefore, if the systolic pressure and the pulse pressure under the same condition vary proportionately, that is, both increase, then the cause lies in the heart, while if in reverse direction or if not proportionately, then the cause lies at least in part in changes in vascular tone. The total cardiac output in a given time also bears a relation to the number of systoles per minute.

From these facts it would seem safe to construct an equation: Blood-pressure quotient = pulse pressure \div systolic pressure = normally about 0.3.

The S. D. R. Index or Energy Index.—Barach¹ bases his assertions in determining the energy index upon the assumption that systole gives us the energy factor in the heart and diastole the energy in the peripheral resistance. From the pulse rate we know how many pressure impulses to each minute there are in the arterial tree. For example if the systolic pressure is 120, the diastolic pressure 70 and pulse rate 72 per minute, the exertion in one minute would be:

In systole..... 120 mm. Hg. \times 72 = 8,640 mm. Hg.

In diastole..... 70 mm. Hg. \times 72 = 5,040 mm. Hg.

In both..... 190 mm. Hg. \times 72 = 13,680 mm. Hg.

If the assumption of Barach is correct, it follows that the knowledge of the systolic pressure alone gives but a partial clue to what is going on in the circulation. In an extensive series of observations by Barach upon the blood-pressure of healthy young men, this contention seems to be proved.

The conclusion reached is that the highest normal energy index is close to 20,000 mm. Hg. per minute. In a series of ten cases of recognized cardiovascular and renal involvement, five of which afterward had apoplectic strokes, the figures of the total energy or the S. D. R. index were all above 20,000, the highest being 50,400. Barach concludes his article by stating that we may obtain by this indication a definite idea of the cardio-

¹ *Jour. A. M. A.*, 1914, Vol. lxii, 7, p. 525.

vascular energy expanded and the strain under which the heart and blood-vessels are laboring.

The Diastolic Index.—MacWilliams and Melvin¹ in taking blood-pressure observations, find it desirable to determine what they call the diastolic index. This is based upon the discovery that in certain cardiovascular cases there was a decided difference in the diastolic pressure, and consequently in the pulse pressure, when the reading was made on the way up from that made on the way down, and that this finding gave valuable data as to the tendency of the case to vasomotor instability. They found in some cases a difference as great as 20 mm. Hg. between the up and down diastolic readings.

¹ J. A. MacWilliams and G. S. Melvin, *Brit. Med. Jour.*, Nov. 7, 1914, 2818.

CHAPTER VIII

VENOUS AND CAPILLARY BLOOD-PRESSURE

While the pioneers in the study of blood-pressure did not neglect the study of capillary blood-pressure, they failed to attach any special importance to this observation, probably because at that time the dynamics of the circulation were but little understood and also because of these crude methods for estimating capillary blood-pressure.

Regarding the physiology of capillary blood-pressure, we know that, in comparison with the pressure within the arterial tree, capillary pressure is low, and the flow uniform and continuous owing to the length of the conducting vessels, which, together with arterial elasticity and ramification, absorb the rhythmic impulses of the heart.

Conditions Influencing Capillary Pressure.—The anatomic situation of the capillaries places them in a position where alterations in their pressure, may be affected by both arterial and venous pressure. Further, the superficial location of large areas of the capillaries subjects them to the influence of external agencies.

A high venous pressure, by impeding the free flow of blood from the capillaries into the veins, will result in an increase in capillary pressure. The reverse is also true. Capillary pressure is influenced by the proximity of this system to the larger venous trunks, and again by the character of the tissues in which the capillaries are located,

the capillary pressure being higher in dense firm tissues than in soft areolar tissues.

The relative importance of capillary blood-pressure has until recently been rather underestimated, although now it is receiving more deserved attention, because we more fully realize the close relation between the pressure in the smallest arterioles and in the true capillaries, and the maintenance of normal peripheral resistance (Chapter II, page 45) which, as has been shown, is the prime factor, excepting the heart itself, in maintaining normal systolic and diastolic pressure.

The actual pressure in the capillaries stands midway between that of the arteries and that of the veins, but it usually more closely approximates the venous than the arterial pressure.

Methods of Measuring Capillary Pressure.—Various means have been devised for demonstrating capillary blood-pressure, the simplest being to press upon a superficial area with a clean microscope slide until whitening of the tissues beneath occurs. The degree of pressure required to accomplish this decoloration will roughly measure the capillary pressure. Formerly a means of applying graduated weights was employed to some extent, but this has been discarded as uncertain and unreliable.

The most practical improvement in apparatus for the measurement of capillary blood-pressure was made by Adolf Basler and by Warren Lombard¹ who separately made devices for this purpose. The instrument of Basler, the "Ochrometer" appears to be the most practical and productive of most accurate results. Its important features

¹ *Zentralbl. f. Physiol.*, 1911, xxv, p. 157.

briefly are: two small tin compartments, the tops of which consist of clear glass windows, arranged for the introduction of two adjacent fingers of the subject to be tested. One of the compartments contains a very thin-walled small rubber balloon which may be so inflated that it will subject the finger to a very delicate pressure, which pressure is measured in millimeters of mercury. The pressure in the balloon is gradually raised until slight blanching of the finger occurs, this point being the measure of capillary blood-pressure. A reading device composed of branching tubes and prisms allows the two fingers to be observed simultaneously for purposes of comparison. This is simply a refinement of the older methods for compressing and measuring the amount of pressure necessary to produce change in color in the part under pressure.

The Normal Capillary Pressure and Its Modifications.—Employing Basler's instrument, Landerer¹ found that the normal capillary pressure varied between 17 and 25 mm. of mercury, although it may at times rise as high as 70 (in firm tissue and branches of arterioles). The figures given in the "American Text-book of Physiology" are between 24 and 54 mm.

Landerer also made an effort to itemize those diseases in which the capillary pressure was either lowered, elevated or unchanged. His results were generally so variable that they possess very little clinical value, the exception being in cases of high arterial pressure, when he found a more or less constant reduction in capillary tension. This was to be expected, if we believe that high arterial pressure is due, in part at least, to arterial and arteriole tightening or narrow-

¹ Rudolph Landerer, *Zeitsch. f. klin. Med.*, 78, Nos. 1 and 2.

ing, which occurs only at the expense of capillary blood-supply.

The application of cold by reducing the bath temperature by 10° or 15° C. diminishes capillary pressure; with hot baths the results may or may not be the reverse.

This author logically suggests that the condition of the arterioles is far more important in determining capillary blood-pressure than is the arterial pressure; for, if the arterioles are contracted, the capillaries receive a scant supply of blood and the resulting pressure naturally is lowered, from which we may conclude that the most important factor affecting capillary blood-pressure is the condition of the arterioles.

Federn takes the same stand, and states that since 1894 he has been endeavoring to perfect a method which would be easily applied, and which would be accurate in determining the systolic pressure in the smaller vessels, which would be a more accurate means of determining changes in the arteriolar and capillary pressures, than the usual method of compressing large trunks. In his "Optic Estimation of Blood-pressure"¹ he describes a method of measuring blood-pressure in a small artery over the tibia, and in the superior anterior intercostal artery by means of a von Basch compressor connected to a sphygmomanometer, the effect of compression being shown by a straw index fastened to a disk of paper or cork (5.6 mm. in size) and pasted to the skin at the point where the pulse is felt. He states that this method eliminates alterations in pressure due to compression ischemia of a large area, gives accurate readings and is the best means of estimating

¹ *Berl. klin. Wochen.*, Mar. 30, 1914, 21, 13.

the functional capacity of the heart as it gives an indication of the pressure in the capillary system.

VENOUS BLOOD-PRESSURE

General Considerations.—Despite the close relation which must exist between the blood-pressure in the large veins and the proper filling of the auricular chamber, and through it the capacity of the ventricular output, which is the direct source of arterial pressure, there have been but few observations and experiments which are sufficiently reliable to be depended upon for clinical deductions.

Methods of Measuring Venous Pressure.—The direct canular method of Moritz and von Tabora¹ is undoubtedly most accurate and, at the hands of the originators, has not been followed by any untoward results. This method could be used experimentally, though it is hardly available in the consulting room.

Other methods have appeared from time to time since 1900, but have failed of general adoption because of fundamental errors in the apparatus.

Two recent instruments (Hooker and Eyster, and Howell) employing air as the medium of pressure, and recording the findings in centimeters or millimeters of water, are probably, at this writing, the best instrumental means for the measurement of venous pressure.

The instrument of Hooker and Eyster² is a modification and improvement on that of von Recklinghausen. It is composed of an aluminum form, shaped to fit the forearm;

¹ F. Moritz and D. v. Tabora, *Deutsch. Arch. f. klin. Med.*, xeviii, No. 4, p. 475.

² *Johns Hopkins Hos. Rep.*, 1909, xix.

partly closed on the arm side by a thin sheet of rubber-dam having a central rectangular opening, and hermetically closed above by a glass window. When applied, the opening in the rubber-dam is placed over a large vein, when by suitable means the pressure within the chamber is increased until the vein collapses. The amount of air required to accomplish this is measured in centimeters of water by a manometer connected with it.

The apparatus of Howell¹ operates upon a different principle. It employs two cups, each connected to a separate water manometer. One cup is applied to the arm and the other to the forearm, the forearm cup being of very thin rubber. The latter is inflated until it fits snugly without exerting pressure (not more than 1 to 3 cm. of water). The upper cuff is then slowly inflated until the manometer connected with the lower cuff shows a rise. This rise shows compression of the veins in the arms and is incident to an increase in volume in the forearm. At this moment the venous pressure reading is made. It will be noted that the degree of pressure originally placed in the lower cuff will to some extent affect the pressure reading, since the higher the pressure over the forearm, the higher will be the venous pressure recorded.

A simple method, ascribed to Gartner, is described in detail and highly recommended by Oliver² and by Brunton,³ for measuring variations in venous pressure without the aid of a sphygmomanometer. This method requires nothing but a foot rule or measuring tape, the veins of the

¹ A. A. Howell, *Arch. Int. Med.*, February, 1912, ix, No. 2, p. 148.

² G. Oliver, *Quart. Jour. of Med. Oxford*, October, 1907.

³ L. Brunton, "Therapeutics of the Circulation," P. Blakiston's Sons, 1908, p. 84.

subject being utilized as an indicator or manometer. If the veins on the dorsum of the hand are sufficiently visible, we find that when the hand is held in a vertical position with the fingers extended, and is slowly raised, the veins at a certain height above the level of the apex of the heart can be seen to collapse rather suddenly.

Oliver states that at the moment of collapse of the veins the blood-pressure within them is practically nil, being balanced, as it were, by the force of gravity. We may, therefore, express this force, annulling the hydrostatic rise of the blood, in millimeters of mercury. This may be done by making a very simple calculation. If we take the average specific gravity of the blood as 1.060 and that of mercury as 13,570, the 25.5 mm. of blood contained in 1 in., will represent 1.985 mm. Hg. (or approximately 2 mm. Hg.); therefore, if we multiply by 2 the number of inches above the level of the apex of the heart when the veins collapse, we ascertain in millimeters of mercury the venous pressure. According to Oliver, this simple method of measuring the venous pressure affords uniform results and is definite and delicate as it enables one to discriminate between differences of 1 mm. It is important to see that the pressure is not artificially raised. This may occur through nervous perturbation or by obstruction of the venous flow by tight clothing.

In making any observations upon the veins, the arm should be maintained in a position such that the point of pressure is about at the level of the heart, irrespective of the position of the patient. If this detail is not regarded, the force of gravity will enter in and invalidate the findings.

Venous pressure readings are also influenced by the

surrounding temperature and by the thickness of the skin over the vein which is being tested, and also by the prominence of the superficial veins and the presence or absence of phlebosclerosis (said to be of frequent occurrence by Hoobler and Eyster) and edema.

OUR PRESENT KNOWLEDGE OF THE PHYSICS AND PHYSIOLOGY OF VENOUS BLOOD-PRESSURES

Regarding the variations of pressure in different parts of the venous system very little is definitely known, some authorities believing that there exists a negative pressure in the great veins near the heart while others contend that a positive pressure within the venæ cavæ is essential to normal auricular function.

Until quite recently we have assumed that venous pressure passively responds to changes in the peripheral resistance and that it rises and falls inversely with the pressure within the arterial tree. There is now a growing belief supported in part by experimental work (some performed as early as 1890) on both men and animals, that the pressure in the venous side, may be and probably is, dominated by a special nervous mechanism. For we now know that the veins are supplied with motor nerves and it has been shown that the veins respond to epinephrin by constricting¹ while Crawford and Twombly² have shown, in addition to this action, that marked constriction of the vein occurs when subjected to a solution of epinephrin of the strength of 1 to 60,000, even when entirely freed from vasomotor control.

¹ Dunn and Chavasse, *Proc. Royal Society*, London, lxxxvi, 1912.

² A. C. Crawford and M. M. Twombly, *N. Y. Med. Jour.*, Aug. 16, 1913.

Capps and Mathews¹ have made a careful study of the subject of venous pressure and have correlated the available experimental and clinical data in substantiation of the value of further studies in venous pressure.

Mall² in 1892 demonstrated that the portal vein is controlled by constrictor fibers from the splanchnic nerves, and considerable proof has been offered by Roy and Sherrington³ to show that the systemic veins also are supplied by vasomotor nerves. Other valuable contributions to our knowledge of venous pressure have been made by Frey,⁴ von Basch,⁵ Bayliss and Starling,⁶ and Sewall.⁷

On the clinical side, investigations are meager as yet. Calvert⁸ called attention to the value of a rising venous pressure as a danger signal in pleural effusions. Hooker and Eyster⁹ carried out a series of observations on venous pressure changes in various diseases of the heart without conclusive results.

That the veins, like the arteries, have the power to maintain their blood-pressure within wide limits was emphasized by Moritz and Tabora¹⁰ who found that it was necessary to withdraw over 500 c.c. of blood by venesection in a human being, before any appreciable fall in the venous pressure occurred.

¹ J. A. Capps and S. A. Mathews, *Jour. A. M. A.*, Aug. 9, 1913, vol. lxi, No. 6, p. 388.

² *Arch. f. Physiol.*, 1892, p. 409.

³ *Jour. Physiol.*, 1890, xi, 85.

⁴ *Deutsch. Arch. f. klin. Med.*, 1902, lxxiii, 511.

⁵ *Wien. med. Presse*, 1904, xx, 962.

⁶ *Jour. Physiol.*, 1894, xvi, 159.

⁷ Henry Sewall, *Jour. A. M. A.*, Oct. 20, 1906, p. 1279.

⁸ *Bull. Johns Hopkins Hos.*, February, 1908.

⁹ *Bull. Johns Hopkins Hos.*, 1908, p. 274.

¹⁰ *Verhandl. d. Ver. d. inn. Med. Berlin*, 1909, p. 379.

Yandell Henderson¹ has advanced the plausible hypothesis that the function of the venopressor (constricting) mechanism is essential to maintain an optimum feeding pressure to the heart. This theory is further substantiated by the newer researches of Hooker² on venous blood-pressure in man. He finds that normal venous pressure is independent of the ordinary changes in peripheral arterial resistance and that the capacity of the veins may vary without affecting the internal pressure.

Factors Influencing Venous Pressure.—*Effect of Posture on Venous Pressure.*—Change of posture from the erect to the horizontal causes a fall in venous pressure, while change of posture from the horizontal to the erect causes a rise in venous pressure. In the extensive researches of Barach and Marks³ these changes uniformly occurred.

The *position of the part* in relation to the heart is an important, if not the most important factor determining venous pressure in the normal person. Although we find that the veins of the feet do not exhibit the degree of pressure as compared with the arm, that would be expected if calculated on the basis of position (von Recklinghausen).

Exercise either local or general causes a slight rise (to about twice the normal pressure); the same may result from sudden change in external temperature. Actual arterial pressure does not affect the venous pressure nearly as much as does general cardiac decompensation and failure of the right heart. The smaller capillaries are probably more dependent for their changes, upon venous pressure than upon arterial pressure.

¹ Henderson and Barringer, *Am. Jour. Physiol.*, 1913, xxxi, 352.

² *Amer. Jour. Physiol.*, 1914, xxxii.

³ *Arch. Int. Med.*, May 15, 1913, No. 5.

Respiratory Variation.—There is a respiratory variation occurring in the external jugular amounting to from 3 to 4 cm. of water.

Defective cardiac action, particularly that of the right heart results in venous stasis which is accompanied by a rise in venous pressure.

Myocardial degeneration is accompanied by a marked rise in venous pressure which may exceed 25 cm. of water at the cardiac level.

Intravenous Injection.—This procedure causes a temporary increase in venous pressure which, in proportion, affects venous more than it does arterial pressure.

External Compression.—Anything which generally compresses the veins in any unit of the body, as an extremity or the abdomen, will increase venous pressure. This fact is important in connection with the care of chronic cardiac decompensation and this knowledge may be employed to prevent syncope after tapping.

Diurnal Rhythm.—According to Hooker¹ the venous pressure in man exhibits a diurnal rhythm, rising throughout the day from 10 to 20 cm. of water and falling again during the night. Thus his average figures are by day 15 cm. of water, at night during sleep 7 to 8.

Relation of Pulse Pressure to Venous Pressure.—The tendency for both pulse pressure and venous pressure is to increase simultaneously in cardiac cases, irrespective of the lesion.

The Normal Venous Pressure.—As may be expected from the preceding and from the marked influences of gravity, posture and other ever-present factors, there are as yet no

¹ *Am. Jour. Physiol.*, 1913, xxxii, 356.

very narrow limits which may be considered as determining normal venous pressures. That the figures given below are clinically satisfactory is shown by the work of Hooker and Eyster¹ who studied venous pressure in the facial vein and the external jugular in dogs taking simultaneous observations by the direct method of placing a canula in the facial vein and studying the external jugular by the indirect method and by comparison, obtained variations rarely exceeding one (1) cm. of water.

Average Venous Pressure				
Normal			Cardiovascular	
	Av.	Variations	Av.	Variations
Howell.....	7.6 cm.	4 to 13 cm.	13.9 cm.	7 to 25 cm.
Hooker } Eyster }	8.0 cm.			
Moritz and v. Tabora.		4 to 8 cm.	Highest 32 cm.	
Barach and Marks....	11.2 cm.	{ Standing 8 to 18 cm. Horizontal 3.5 to 11 cm.		

Clinical Importance of Venous Pressure.—From our present knowledge of the physiology of the circulation and of its pathologic change in cardiac decompensation, there is little doubt that the venous pressure plays an important part in the proper filling of the heart chambers and that a normal venous pressure at the level of the heart is essential to a normal cardiac output. This is shown to be true at least in part by the fact that pulse pressure and venous pressure vary simultaneously in health (see above), also by the evident over-distention of the veins in cardiac decompensation from whatever cause, and the marked, and sometimes spectacular, relief afforded by appropriate and timely

¹ *Loc. cit.*

venesection. These facts all indicate that venous pressure, when high, is at least one factor which interferes with the normal action of the auricle, rendering it, for the time, powerless to functionate properly until relieved by a *vigorous* reduction in venous pressure. In this connection, Bishop has pointed out that in determining the seriousness of arterial hypotension, the chief factor is the approximation of venous and arterial pressure, and that a low pressure need not necessarily be serious, *per se*, unless the venous pressure is abnormally high, and further that it is this altered relation between the normal venous and arterial pressures that determines the seriousness of general venous congestion.

Concerning pulmonary venous pressure, we have no method of clinical value by which the degree and changes in pulmonary venous pressure may be determined. Clinical phenomena, particularly the wetness or dryness of the lungs in cardiac cases will determine this.

Effects of Drugs on Venous Tension.—A carefully planned and rigidly checked series of experiments by Capps and Mathews¹ are worthy of consideration as they comprise almost our sole accurate data. They employed the method of Moritz and Tabora (see page 146). The summary of their results is as follows:

Digitalis Group.—*Digitalis* $\frac{1}{130}$ to $\frac{1}{120}$ gr., *digipuratum* 1 c.c., *strophanthin* $\frac{1}{120}$ to $\frac{1}{60}$ gr., all used intravenously. No effect on venous pressure.

Epinephrin.—1.2 to 10 min. 1-1000 sol. diluted in 20 c.c. salt solution. Small doses no effect, large doses rise in venous pressure from 10 to 80 mm. H_2O .

¹ *Loc. cit.*

Pituitrin.—1 c.c. 10 per cent. sol. Same as epinephrin but less marked effect.

Caffein.—Caffein sodium benzoate $\frac{1}{2}$ to 2 gr., no effect.

Strychnin Sulphate.—No effect except in toxic quantities, then a rapid rise in venous pressure.

The Nitrites.—Amyl nitrate and nitro-glycerin $\frac{1}{150}$ to $\frac{1}{50}$ gr. caused a decided fall in venous pressure.

Morphine.— $\frac{1}{8}$ to $\frac{1}{6}$ gr. No effect on venous pressure. $\frac{1}{4}$ to $\frac{1}{2}$ gr. Slight fall in venous pressure.

Alcohol.—25 to 30 per cent. strength 10 to 50 c.c. in amount. Small doses no effect, large doses rise in venous pressure. This rise was in proportion to the degree of disturbance of heart action.

CHAPTER IX

CLIMATOLOGIC AND RACIAL INFLUENCES

Absolute and Relative Blood-pressure.—Throughout the discussion of this subject, the relative blood-pressure only has been considered, that is, the degree of arterial pressure over and above barometric pressure at the time of the observation. This has, from a clinical standpoint, been clearly demonstrated to be the important factor, as ordinary studies do not involve a determination of the absolute blood-pressure, *i.e.*, the systolic pressure plus the actually determined barometric pressure at the time of the observation. It may be stated in passing that changes in atmospheric pressure are accompanied by approximately equal changes in the absolute blood-pressure, usually in the same direction.

Altitude.—In approaching the subject of the effect of altitude on blood-pressure and pulse rate, a sharp line must be drawn between the influence of changes in altitude (atmospheric pressure) upon normals and other individuals, particularly the tuberculous; otherwise confusion will surely follow, because the great bulk of clinical data demonstrates that altitude affects normal and pathologic individuals differently.

Effect of High Altitude on Healthy Individuals.—Gardner and Hoagland,¹ at an altitude of 6000 ft., meas-

¹ Gardner and Hoagland, *Trans. Amer. Climat. Assn.*, 1905.

ured large numbers of normals who had lived in Colorado for more than a year, and concluded that the average blood-pressure was slightly lower than at the sea level, although prolonged residence at that altitude does not materially affect blood-pressure.

Experiments showed that an ascent from 6000 to 14,000 lowered pressure and increased pulse rate. The fall was apparently a permanent one, although the fall which immediately follows the change in altitude is temporarily greater than is the case after the individual becomes accustomed to the alteration in altitude.

Smith at Ft. Stanton¹ (6200 ft.) states that, "It has been scientifically established that blood-pressure is lowered with increased altitude."

Pomeroy² in order to determine the degree of variation in the systolic and diastolic blood-pressure, caused by changes of altitude, averaged the observations of eighteen investigators, dating from 1878 up to the date of this writing and including himself. He found that the fall in systolic blood-pressure ranged between 1 and 22 mm. Hg., and the diastolic fall was between 1 and 11 mm. Hg.

The effect of sudden changes in altitude in the upward direction results usually in an increase in systolic pressure, an increase in diastolic pressure, with very little if any significant change in pulse pressure. These changes were accompanied by nose-bleed and headache.³ In another group which were largely pathologic, no such changes were noted.

In order to determine the effect of increased atmospheric

¹ *Reprint* No. 51, Public Health Rep., P. H. and M. H. Service.

² *Int. Med. Jour.*, Vol. xviii, p. 731.

³ Personal communication from Dr. Harley Stamp.

pressure upon the blood-pressure and pulse rate, Brooks¹ studied seventy-five compressed-air workers, when contrary to the findings of earlier investigators, he found that only rarely did there occur a marked rise in arterial pressure, even in workers subjected to a pressure of plus 31 lb., as in no cases examined by him was blood-pressure or pulse rate materially altered. Further observations, if in accord with these, would be of value in demonstrating the degree of danger involving cussion workers who are subjects of cardiovascular and renal impairments.

Blood-pressure in Deep Mines.—Investigations after descent into the Butte mines, to a depth of 2600 ft. below the surface, was made in an effort to determine the effect of this change upon blood-pressure. The usual effect was found to be a fall in both systolic and diastolic pressure with a primary increase in pulse pressure, which tended in a short time to return toward normal values.²

Schneider and Hedblom³ present a very concise and accurate summary of present knowledge bearing on this point.

1. A considerable elevation in altitude tends to lower systolic and diastolic blood-pressure and to increase the heart rate.

2. The fall of systolic pressure is slightly greater and more certain to occur than the fall of diastolic pressure.

3. A rise in diastolic pressure occurs in some individuals.

4. The influence of such factors as psychic states, eating and exercise may obscure the findings.

¹ H. Brooks, *Med. Rec.*, May 25, 1907.

² Personal communication from Dr. Harley Stamp.

³ *Am. Jour. Physiol.*, Vol. xxiii, No. 2.

5. The fall in blood-pressure and increase in heart rate are more marked in the early part of stay in high altitudes.

6. With prolonged stay in high altitudes the heart rate probably returns more nearly to normal than the blood-pressure of all individuals.

7. High altitudes do not affect in the same degree all individuals.

8. Small elevations in altitude do not materially influence blood-pressure.

9. Those individuals most affected by high altitudes seem to sustain the greater fall in systolic blood-pressure and the greater acceleration in heart rate.

10. The heat of the summer season probably accelerates the pulse rate.

11. Moderate variations in altitude are not of themselves inimical to residence or physical activity in persons having definite cardiovascular or nephritic lesions.

Tuberculosis.—LeRoy S. Peters, pointed out in 1908¹ that altitude usually caused a rise in blood-pressure in the tuberculous. He made his observations at an altitude of 6000 ft. (For effect of tuberculosis on blood-pressure see Chapter XIV.)

Bullock² confirms the observations of Peters. The blood-pressure raising effect of altitude on persons suffering from pulmonary tuberculosis appears to be of distinct advantage to the patient, as it directly combats the blood-pressure reducing acting of tuberculotoxins by altering metabolism, modifying and stimulating tissue change, and aiding elimination.

¹ *Arch. Int. Med.*, August, 1908.

² *Jour. A. M. A.*, June 19, 1909.

B. R. Hooker¹ shows, in his reports of respiratory cases, that placing patients in the open air increased the blood-pressure from 5 to 10 mm.

Influence of Climate.—Weston P. Chamberlain has recently reported in the *Philippine Journal of Science*² an exhaustive study of the effect of climate and race upon the normal average blood-pressure readings. The study is based upon 6128 blood-pressure observations on 1042 white men and 552 Filipinos all in good health and ranging in age from twenty to forty years. The average systolic pressure of 5368 readings on 992 persons, was 115.6 mm. and the pulse rate taken simultaneously averaged 81 beats per minute. The average age was 26.6 years. Comparing this average with that of Woley (see page 42)

Chamberlain's Table.—Average systolic blood-pressures and pulse rates, based on 5368 observations of each which were made on 992 American soldiers serving in the Philippines; arranged according to age. (12.5-cm. armlet.)

Age period, years	Average age, years	Number of men showing pressures from—							Total number of men	Average pressure	Average pulse rate
		91 to 100 mm.	101 to 110 mm.	111 to 120 mm.	121 to 130 mm.	131 to 140 mm.	141 to 150 mm.	151 to 160 mm.			
18 to 20	19.4	1	12	13	8	1	1	36	Mm. 115.0	78
20 to 25	22.8	32	156	165	87	22	5	2	469	114.3	82
25 to 30	27.2	16	73	108	70	13	3	3	286	115.9	81
30 to 35	32.6	2	34	42	23	7	1	109	116.7	80
35 to 40	37.5	9	24	14	8	3	58	120.5	81
Over 40	43.1	2	3	17	7	2	2	1	34	119.6	79
Totals or averages	26.6	53	287	369	209	53	15	6	992	115.6	81

¹ *Med. Rec.*, Jan. 28, 1911.

² December, 1911, Vol. vi, No. 6, Sec. B.

it is found to be 7 mm. lower and compared to Bachman¹ 3 mm. lower. While the pulse rate in Chamberlain's series was 9 beats per minute above the average accepted as normal in temperate climates for all ages. He also found that the blood-pressure has a tendency to be lower than the averages given above, during the first three months stay in tropical climates.

Racial Influence on Blood-pressure.—Chamberlain also reported² a series of observations conducted to determine the effect of race upon average systolic blood-pressure and obtained the following result:

Average blood-pressure of 100 Filipino scouts, 115.0

Average blood-pressure of 100 Philippine soldiers, 115.9

and states that "we may, therefore, conclude that the mean blood-pressure for Filipinos of from fifteen to forty years of age (average age twenty-five years) is 115 to 116 mm. and that it does not differ from the pressure at the same age for Americans residing in the Philippines."

Normal Blood-pressure Studies in the American Indian.—The investigations of Dr. Harley Stamp who was attached to an archæological exposition sent out by the University of Pennsylvania headed by Dr. Frank G. Speck, have brought out some very interesting points in connection with the comparative study of blood-pressure. These observations for the most part were carried on in the winter of 1913-14 to the number of many thousand upon fifty-seven tribes of American Indians, and often at from 20° to 30° below zero. Another group of the observations

¹ *N. Y. Med. Jour.*, 1911.

² *Loc. cit.*

was made at the Carlisle Indian School, thereby giving some comparative data as to the effect of civilization upon blood-pressure in the American aborigine. In summarizing these observations it may be said that the effects of exercise probably subside more quickly in the Caucasian than in the American Indian. "The effects of low temperatures were undoubtedly very noticeable; the effects also of the habits, not only in reference to their food but also to the lack of sanitary requirements, even of the most primitive kind."

These observations were all made by accepting the first point as the systolic and the fifth point as the diastolic pressure criterion. The individual ages ran from two months to 103 years, and the Indians were of all sizes, the same as found in any Caucasian community. There were among them approximately the same proportion of good muscular development.

The diurnal and periodic changes showed no differences from similar observations upon Caucasians, while emotion causes a far greater variation in the Indian than in his white brother.

This investigator is inclined to believe that the individual inclines to become acclimated to low temperatures, which causes the variation to become less as the low temperatures are endured. There was no difference noted in the effect of postural changes, or of alcoholic indulgence. In the use of tobacco there is a marked difference in the effect on the two peoples. This seems to be due to excessive smoking and to the use of "doped" tobacco by the Indian. Much of this is taken in the form of snuff. The tea habit seems well formed and the record

is made of a guide who drank forty-two cups of strong tea a day.

The normal age charts of blood-pressure records upon the American Indian differ but slightly from those of the white except that there seems to be a uniform tendency to reduced blood-pressure at or about the forty-fifth year.

CHAPTER X

PHYSICAL FITNESS, EXERCISE AND ATHLETICS THEIR EFFECT UPON AND RELATION TO BLOOD-PRESSURE

General Considerations.—The physiology and pathology of exercise are chapters in medicine, which have just begun and in which there is much to be written. A number of facts, however, have been established which permit of certain generalizations, this being particularly true of our knowledge of the response of the heart to changes in the circulation induced by muscular activity, and it is generally recognized that no one should be permitted to engage in exhausting sports or unusual physical exertion, who does not possess an effective cardiovascular system, which will insure him against possible untoward results from the extraordinary effort.

Thus we find that a careful and thorough physical examination, with special reference to the condition of the heart, circulation and kidneys made by a competent medical examiner, has become a recognized practice in most gymnasia and athletic institutions, where youths train and engage in competitive athletics.

The indulgence in athletics as a means of preserving health and physical well-being, and the rational employment of special exercises as one of the devices of physical therapy now demand of the active physician a comprehensive under-

standing of certain physiologic phenomena and the consequences following various forms of muscular activity.

CARDIAC EFFICIENCY IN RELATION TO EXERCISE

As it is clearly evident that the response of the cardiovascular system to muscular effort depends largely upon the integrity of the musculature of the heart, it follows that in approaching this subject the dividing line must be sharply drawn between individuals having abnormalities of the heart and arterial systems and those whose circulatory apparatus is intact.

Extreme caution should be exercised before permitting active muscular exertion and competitive exercise to individuals who are known to have cardiovascular and renal defects, and when permitted always should be under constant competent supervision. This does not mean that the presence of cardiac, renal and vascular defects, *per se*, preclude indulgence in all forms of sports and exercises; indeed the judicious employment of properly supervised and carefully adjusted muscular effort, including certain mild sports, should form a large part of the medical care of persons coming under this group.

Cardiac efficiency or cardiac effectiveness means cardiac muscle integrity, and this in turn implies not only an ability of the heart to supply the normal demands of everyday life, but also an ability to respond to extraordinary demands in such a manner that an adequate circulation will be maintained.

It is believed that the normal heart in youth with the body at rest, does not expend more than one-twelfth of its potential or reserve energy. Under ordinary conditions

of active life, involving moderate work, from one-fourth to one-third of reserve power is called out, while the full capacity is only demanded by the most severe forms of exercise demanding great endurance. This reserve or potential energy is closely related to the demands of the tissues and organs for additional blood. With the body at rest it is estimated that 60 c.c. of blood is expelled from the heart at each systole, at the rate of seventy-two times per minute. Upon demand this volume output may be increased sixfold, the increase in output being partly the result of an increased cardiac rate and partly an increased systolic capacity.

The whole question of cardiac efficiency hinges upon the presence of an adequate potential reserve or factor of safety, and the development of cardiac insufficiency is almost wholly dependent upon the degree of reduction in cardiac reserve as compared with the demands for the expenditure of excess energy. It is upon these two factors, cardiac reserve, and the demand for expenditure of energy that the degree of physical fitness depends.

Concerning the formulation of hard and fast rules, regulations and restrictions, for the guidance and control of individuals with such defects, very little can be said, as every case differs from every other and should be governed by mature judgment, based upon the results of careful physical examination, functional tests and experimental exercises.

Given an evident "pathologic fault," the most practical means at our disposal with which to corroborate and qualify the results of the physical examination are, an analysis of the urine and a study of blood-pressure, especially noting in the latter, variations in systolic, diastolic

and pulse pressures, and also pulse rate under changing conditions of rest and exercise. The so-called functional tests (see Chapter XIX, page 331) should be applied to all except adolescents and youths. A pathologic urinary finding will, of course, be given its proper value, and be collated with the other evidence at hand.

Valvular defects alone have little effect upon normal blood-pressure values, especially in the young, excepting those with aortic regurgitation, and in those past middle life, whose reserve has become reduced and where vascular and renal degenerative changes contribute to the circulatory instability.

Concerning the pure forms of cardiac hypertrophy, such as occur in the absence of valvular lesions or the history of previous disease, Barach¹ is of the opinion that when subjected to strain they differ very little from normal hearts, as in a series of observations on 50 youths he noted only the usual transitory dilatation following overstrain from long-distance running.

Continued indulgence in athletics is generally essential to good health in this class, as exercise tends to delay the onset of degenerative changes which would follow the development of sedentary habits of life.²

The discovery of an abnormal blood-pressure value (see Chapter XVI, page 246, for discussion of causes) while not a cause for eliminating all exercise, will tend to move such individual's age limit forward and so reduce the amount of exercise to be safely indulged.

Arteriosclerosis even without hypertension suggests the

¹ *Jour. A. M. A.*, Oct. 29, 1910, lv, 18, p. 1581.

² Robert E. Coughlin, *Med. Rec.*, Apr. 2, 1910.

same caution, except where the arterial change is purely local and evidently limited to the superficial vessels, as in those accustomed to manual labor without any renal lesion.

For the detection of myocardial degeneration and insufficiency recourse must be had to the functional tests, as advocated by Graüpnér,¹ Boardman Reed,² and others.

Graüpnér and others, who have investigated the question of the rise and fall of systolic pressure during and after exercise, have found that normal hearts, those with well-compensated valvular lesions, and neurotic hearts, respond to exercise by a rise in pressure; whereas myocardial cases either fail to show a rise or respond by a lessened tension varying from 3 to 15 mm.

E. Masing³ advises a careful study of the effect of exercise upon the normal relation between systolic and diastolic pressure, *i.e.*, the effect of exercise upon the pulse pressure. Thus a normal circulatory system will yield a disproportionate rise in systolic pressure as compared with diastolic, thereby producing temporarily an increased pulse pressure, while with a defective circulation, even if there occurs a rise in systolic and diastolic pressures, the two tend to approximate and thus the pulse pressure becomes smaller.

Age in Relation to Athletics.—In order to study the effect of exercise upon the normal individual and the amount of energy expended in relation to blood-pressure variation and to the margin of cardiac safety, Coughlin found it

¹ *Berl. klin.*, 1902, xv, 174.

² *South. Calif. Pract.*, August, 1910.

³ *Deut. Arch. f. klin. Med.*, 1902, lxxiv.

convenient to group the athletic age of man under four divisions,¹ viz.:

1. Early life, including infancy, childhood and youth, up to twenty-one years.
2. Manhood, from twenty to forty years.
3. Middle age, from forty to fifty-five years.
4. Beyond middle life, up to old age.

1. *Early Life to Young Manhood*.—Of first importance at the approach of what may be called the threshold of athletic life is the determination of the presence or absence of cardiovascular or renal abnormalities which might subject the individual to grave danger if not discovered before active athletics are indulged in.

Parents of growing children are beginning to appreciate the value of more definite information concerning their children's physical condition, both to determine the presence of possible abnormalities, and also to avoid the development of future physical defects and weaknesses, through a knowledge of the character and amount of exertion that may be safely indulged in.

The problem is frequently brought to the physician for solution, by a question somewhat like the following: "Doctor, my boys are going to boarding school this fall and I am anxious to know whether their physical condition is such that they may indulge in track work, football, basketball, etc."

The solution of this problem is not so easy as it appears and to give a definite reply is to shoulder a great responsibility.

¹ R. E. Coughlin, *Med. Rec.*, Apr. 2, 1910.

Cardiac efficiency and physical fitness are not necessarily synonymous, for a poor physique with a normal circulation may, if subjected to ill-advised exertion, rapidly develop cardiovascular deficiency, while under proper guidance the same individual might gradually develop into a hardy athlete.

The demonstration of cardiovalvular defects, should place one upon their guard, and the character and extent of exertion permitted will be determined by the particular lesion discovered, the degree of compensation present and the age of the individual. Aortic lesions of any kind and obstructive lesions of any valve are more serious than a mitral regurgitation or a leakage due to a relative insufficiency. All such cases should have their reserve and muscular efficiency determined by appropriate study and tests (see Chapter XIX, page 331).

In giving advice to the normal individual in this class, the age, muscular development and general build, heredity, past history, and personal idiosyncrasy must be taken into consideration.

In individuals where any question is raised, much may be done by a progressively graduated entrance into athletics, in which the child or youth is led gradually from mild to more active exercises, and so to the more exhaustive sports as his ability to withstand strain is demonstrated.

In regard to the character of exercise that may safely be undertaken in boys' schools, an editorial in *The Hospital* for April 3, 1909, pointed out that neither age nor distance (track athletics) are in any way an exact criterion of the strain inflicted upon any given boy in any given race, and that the quarter-mile is often far more exhausting, for most

boys, than longer distances. This review also condemns the plan of running boys of all ages in the same heats or over the same distances; because, to determine the classes by age may mean that a stocky, well-developed boy capable of any exertion may be sent into a junior division so setting smaller boys a hot pace for a short run, while an overgrown but less precocious boy, perhaps only a month older, may be made to compete with the most athletic of his fellows over a long distance.

An abundant and somewhat contradictory literature has grown up concerning the effect of exercise on the heart. Recently Shumacher and Middleton¹ have given a brief résumé of some of the more important work. These authors incline to the view that athletic training at first leads to physiologic hypertrophy, but when prolonged and markedly severe, it usually leads to hypertrophy plus dilation of a variable degree, frequently marked by valvular insufficiency, and while they admit that such hearts may be efficient even in severe athletic contests, they nevertheless believe that the so-called "athletic heart" is a distinct disadvantage to normal human activities. On the other hand, James Mackenzie² seriously doubts the existence of any such pathologic condition and maintains that "the evidence on which such heart impairment was based, was those manifestations of murmurs or irregularities which my experience has shown to be perfectly consistent with a healthy heart." Cabot³ studied 600 consecutive cases with signs of cardiac insufficiency, for the purpose of

¹ L. Shumacher and W. S. Middleton, *Jour. A. M. A.*, lxii, p. 1136, 1914.

² *Brit. Med. Jour.*, 11, p. 1697, 1912.

³ R. C. Cabot, *Jour. A. M. A.*, Vol. lxiii, p. 1461, 1914.

determining the etiologic factor and in no case was he able to trace the cardiac impairment to athletics or to immoderate exercise.

Lee, Dodd and Young¹ report a systematic study of athletes of different ages, made for the purpose of determining the etiologic relation of athletics and strenuous exercise (rowing) to cardiac size and efficiency.

They divided their men into three groups:

1. Freshmen.
2. Candidates for first university crew.
3. Graduates, who averaged over ten years of rowing.

Preliminary physical examinations showed all members of all groups to be free of cardiac irregularity, kidney involvement or cardiac enlargement as demonstrated by the usual methods of percussion and auscultation.

They found in group 1 an average heart width of 13 cm., varying from 11.5 to 14.2 cm. In group 2 an average cardiac width of 13.85 cm., varying between 12.7 to 15.1 cm. In group 3 an average cardiac width of 14.04 cm., with variations between 11.5 and 15.5 cm. A series of non-athletes showed an average width of 14 cm.

These studies show a surprisingly small difference between any of the groups, particularly between those who had rowed from two to four years and those who had rowed over ten years, this difference being 0.19 cm. or only $\frac{1}{13}$ in. The difference between groups 1 and 2 is fully explained on the basis of age and physical development.

These authors conclude that there is no evidence to show that prolonged participation in rowing under proper super-

¹ Rodger I. Lee, Walter J. Dodd and Edward L. Young, *Boston Med. and Surg. Jour.*, Sept. 30, 1915, clxxiii, 14, p. 499.

vision materially increases the size of the heart which is sound at the beginning.

It may be concluded, therefore, that in youths with normal circulation and kidneys and average muscular development, who have been physically active during the time of their early development, there is very little danger that muscular exercise will permanently or seriously damage the heart, because of the great natural reserve and resilience of this organ and of the arterial coats; but special care should be observed in permitting active exercise after acute illness, particularly the infectious diseases, even when mild, because of the great tendency of these diseases to produce temporary myocardial weakness.

2. *Manhood up to Forty Years.*—Indulgence in athletics between the twentieth and fortieth years in the absence of demonstrable cardiovascular and kidney defects, is fraught with least danger, as the majority of those who continue to be so active, have grown up to it, having followed some degree of athletic training through high school and college and are therefore well prepared for such exertion. On the other hand, those not formerly so inclined are usually too busy attending the urgent demands of modern life to actively indulge in competitive sports; furthermore, degenerative changes are not at this time frequent, so that although a man may be working under high pressure, his cardiac reserve remains sufficient to cope with any emergency. Regarding the probable immediate effects of severe exertion upon trained athletes prior to any contest, more will be said in the section on functional tests.

3. *Middle Age.*—Regarding the ability of the system to withstand the strain incident to strenuous athletics after

forty years of age, little can be said except that, given an individual accustomed to athletic competition, who throughout the greater part of his life has devoted a certain amount of time regularly to such sports, he may be per-

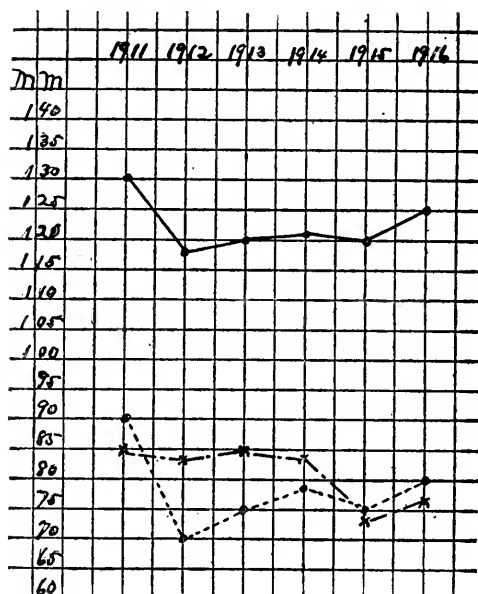


FIG. 36.—No. 26. Male aged sixty-seven. Normal. Has always enjoyed good health except for an occasional attack of neuritis the result of exposure to extremes in temperature. This patient has been under continuous observation for five years and the chart shows a summary of observations for each year. Note the consistently uniform level of both systolic and pulse pressures except for the first year, during which improved hygienic measures were instituted. The urine at no time has shown any abnormalities and the heart has always been adequate even after moderate exercise tests such as ten bending movements and walking up and down stairs.

mitted to continue, so long as physical examination, including blood-pressure tests, fail to bring out cardiac overstrain or cardiovascular and renal degenerative changes (Fig. 36).

Regarding those who would take up active athletics after forty years, much will depend upon the character of the sport and the physical fitness of the individual. Generally speaking a physically normal person may with safety and often with benefit indulge in such sports as cricket, golf, rowing, etc., provided the approach be gradual and sufficient time is consumed in developing proficiency to allow the system to develop a reserve, *i.e.*, the performance of the greatest amount of work with the least expenditure of energy.

Fifty-five Years and Over.—The preceding remarks apply here with even greater rigidity, as there are few who can, with impunity, indulge in any active athletic exercise who have previously led an entirely sedentary or passive life. The degree of activity allowed must be determined in part by the heart, circulation, and kidney condition and in part by the response by the individual to muscular strain while under observation. The demonstration of any defect should preclude all violent exertion and athletic competition.

From the opposite standpoint a few important facts apply to divisions three and four. Thus it is an accepted fact that men over forty do not exercise sufficiently to keep up metabolism and it is equally well known that faulty metabolism is an important, if not the most important, cause of degenerative changes. So that, rather than discourage moderate exercise after forty years, it would seem better to determine carefully the individual's ability to withstand exertion and then judiciously advise systematic exercise, as there is no question that degenerative changes and senility may be greatly delayed by continued athletic

indulgence, particularly in those who have in earlier life been athletically inclined.

The Effect of Exercise upon the Heart, Circulation and Respiration.—The study of the immediate effects of prolonged muscular exertion upon the normal individual has for many years furnished a fruitful field for special investigation.

From a careful review of literature bearing upon this subject, it appears that the earlier conclusions reached by such observers as Karrenstein¹ Gärtner,² Gordon,³ and Moritz⁴ who believed that severe exercise resulted in a fall in systolic blood-pressure, were not correct, and that our views upon this subject must be modified to conform with more recent and accurate studies.

At present the consensus of opinion warrants the following conclusions:

1. Muscular exercise when sufficient to increase cardiac activity is followed by a rise in both systolic, diastolic, blood-pressure and pulse-rate⁵ and in venous pressure.⁶

2. As a rule, there is a greater rise in systolic pressure than in diastolic pressure; therefore, exercise normally increases the pulse pressure, which may be interpreted to indicate an increase in cardiac output as well as an acceleration of its action.^{7,8}

3. The rise in systolic pressure continues to increase

¹ *Amer. Jour. Med. Sci.*, 1912, cxliii.

² *Zeitsch. f. klin. Med.*, 1903, Vol. 1.

³ *Edinburgh Med. Jour.*, 1907, xxii.

⁴ *Deutsch. Arch. f. klin. Med.*, 1903, lxxvii.

⁵ C. S. Lowsley, *Med. Jour. Physiol.*, Mar. 1, 1911.

⁶ Hooker and Wolfsohn, *Am. Jour. Physiol.*, xxv, 24.

⁷ Lowsley, *loc. cit.*

⁸ Masing, *Deutsch. Arch. f. klin. Med.*, 1902, Vol. lxxiv.

after exercise is begun, until a maximum is reached. A period of rest is accompanied by a decline in pressure, which again approaches the maximum rise as the exertion is renewed. The systolic pressure does not return to the normal level for some time after the muscular effort ceases, but will finally fall to below normal, when it constitutes what is known as a "negative phase."¹

4. The extent of the rise in systolic pressure depends on the character of the exercise, upon the individual and his condition. Experimenting with young men upon a stationary bicycle, Lowsley found that the time when the maximum systolic pressure was reached varied from five to twenty-five minutes and that the increase amounted to from 10 to 65 mm. with an average rise in sixteen minutes of 32.7 mm. Apparently the greatest rise occurred when the man was fresh.

5. The rise in the diastolic pressure reaches its maximum either at the same time as the systolic, or a few minutes later. In Lowsley's work cited above, the rise in diastolic pressure varied between zero and 40 mm. while the average in seventeen individuals was 22.9 mm.

The diastolic pressure fluctuates less after the maximum has been reached than does the systolic. The return to normal after exercise is slower but it invariably falls to below normal before the final balance is established.

6. The pulse pressure curve generally follows the systolic, this being so because the systolic pressure fluctuates more than the diastolic.

7. The venous pressure generally rises to a maximum rather rapidly and remains there throughout the period

¹ E. O. Otis, *Am. Jour. Med. Sci.*, Feb. 1, 1912, cxliii, No. 2.

of exercise. This rise rarely amounts to more than 14 cm. of water. If, however, the exercise be not severe and deep regular breathing accompanies the "second wind," then the venous pressure may return to normal even during the exertion. As a rule it falls fairly rapidly to normal after exercise but occasionally remains high for a considerable period (Hooker).

8. During exercise requiring severe strain, with rigid chest and closed glottis, the systolic pressure is increased more than the heart rate, and venous pressure may be exceedingly high.

9. The pulse-rate increases rapidly at first but does not usually attain its maximum as soon as does the blood-pressure. In Lowsley's experiments the average time for reaching the maximum pulse rate was 35.4 minutes, and the average increase in nine experiments was 51 beats per minute. Cook and Pembrey¹ have noted an increase in pulse-rate up to 180 per minute. After the maximum rate has been reached there is not much variation during the period of activity. After cessation of muscular effort, there is a fall to normal and rarely to subnormal,² much less rapid than the fall of blood-pressure. After prolonged, severe exercise, the pulse-rate may remain above normal for a long time and in some cases there may be a secondary rise which appears to be due to a reflex effort to improve the negative phase of low pressure.

The recovery to normal is most rapid in well-trained men whereas in the untrained, severe exercise causes a persistently rapid pulse often irregular, which may remain so for some time.

¹ *Amer. Jour. Physiol.*, 1913, xlv, p. 429.

² Lowsley, *loc. cit.*

10. Since after prolonged exercise the systolic pressure falls more rapidly than the diastolic, there results a period of small pulse pressure. At the time of this small pulse pressure albumin is frequently found in the urine. An albuminuria coincident with low pressure after exercise has been described by Erlanger and Hooker.

11. The more rapid, vigorous, fatiguing and exhausting the exertion, the more probable the subnormal phase and the longer its duration.¹ A subnormal phase frequently occurs if the exercise be short and exhausting, as well as when moderate but continued over a longer period of time. Lowsley also suggests that the duration of the subnormal phase may be taken as a very fair index of the strain on the circulatory system and he has come to believe that a subnormal phase of less than an hour may be considered as safe, while if it persists for more than two hours, it is a sign that the margin of safety has been exceeded.

12. Long distance running and exhausting boat races and other similar forms of exercise are a serious strain upon the heart which is indicated by a prolonged period of subnormal pressure or negative phase.

13. In a hypertrophied heart, the seat of a systolic murmur, not due to valvular lesions, severe athletic contests may cause the murmur to temporarily disappear, or on the other hand, systolic murmurs may appear in a heart in which none were present before the contest (Barach).

14. After prolonged and severe contests, arrhythmia may appear. This is best explained by Shumacher and Middleton as being the result of mechanical injury to the con-

¹ E. O. Otis, *Amer. Jour. Med. Sci.*, February, 1912, cxliii, No. 2.

ducting apparatus; that is, to fibrillary tears in the myocardium.¹

15. By the teleröntgen method, C. S. Williamson² has demonstrated that (a) the normal heart responds to any exercise within its power by a diminution in size; (b) about 50 per cent. of pathologic hearts, which are in good compensation, respond to exercise within their power, by a diminution in size; (c) approximately 50 per cent. of the pathologic hearts, which manifest but a low grade of broken compensation, respond in the same manner by some diminution in size.

Permanent Result of Prolonged Muscular Exercise and Cardiac Overstrain.—There is less argument concerning the ultimate result of over-exertion on the heart than upon the immediate effect of physical exertion. The following points, however, seem to be clearly established:

1. Severe exertion from violent and prolonged efforts may reach a point beyond the capacity of the heart muscle to produce a complete systole. This results in the typical symptoms of acute cardiac dilatation. The prognosis here depends upon whether the heart was previously sound or not.³ The weakened condition of the cardiac muscle is shown after the cessation of effort by its inability to return to its normal size.

2. Infectious diseases especially tend to weaken the myocardium. For this reason, severe athletic contests should be avoided by those suffering with, or convalescent

¹ L. Shumacher and W. S. Middleton, *Jour. A. M. A.*, Apr. 11, 1914, lxii, No. 15, p. 1136.

² *Am. Jour. Med. Sci.*, 1915, cxlix, No. 4.

³ K. Blandenburg, *Med. klin.*, Sept. 3, 1914, x, 37.

from, acute infectious diseases, even often mild cases of tonsillitis and grippe.

3. Immoderate physical efforts lead to hypertrophy of the heart. Athletic training leads first to physiologic hypertrophy, but if prolonged and overdone it leads to hypertrophy plus dilatation. The degree is variable and may frequently lead to marked valvular insufficiency.¹

4. Functionally, the hypertrophied heart, even when dilated and giving distinct evidence of valvular insufficiency may prove more fitted to carry the man through a severe athletic contest than a normal heart would be. On the other hand, acute cardiac dilatation occurs more frequently in athletes and men habituated to severe muscular strain than in average men, and the ultimate effects are more prolonged and severe.²

5. Hearts known to be diseased may respond in the normal manner, as shown by a reduction in size after the cessation of moderate exertion, while hearts that give a distinct systolic murmur may withstand a Marathon race better than hearts which are apparently nearer normal.³

6. There is reason to believe that for normal human activities an "athletic" heart is distinctly disadvantageous.⁴

7. Experience teaches that much good can be secured by careful examination of the heart before and after moderate exercise, of all those who desire to enter severe athletic contests. Failure to obtain a normal reaction should bar anyone from such contests. Even those apparently standing such contests well should have the heart and blood-

¹ Shumacker and Middleton, *loc. cit.*

² Shumacker and Middleton, *loc. cit.*

³ Barach, *loc. cit.*

⁴ Shumacker and Middleton, *loc. cit.*

pressure examined at frequent intervals during training, to detect possible overstrain. Continued overstrain even when slight may weaken a normal heart muscle instead of strengthening it. This appears to be particularly true among young boys (of immature development) who have been found to develop persistent cardiac changes the result of prolonged overstrain.

8. Carefully collated statistics¹ show that athletes die from the following diseases in their order of frequency: cardiac disease, tuberculosis, typhoid fever, pneumonia and Bright's disease.

¹ R. E. Coughlin, *Med. Rec.*, Aug. 8, 1914, lxxxvi, No. 12.

CHAPTER XI

BLOOD-PRESSURE IN CHILDREN

Reference to the normal blood-pressure values in children has already been made in the section devoted to normal blood-pressure and the factors causing its variation, so that further discussion of this portion of the subject is omitted here (see page 115).

In studying the difference between the average pulse pressure in children and in adults, G. S. Melvin and J. R. Murray,¹ employing a Pachon oscillometer with an 8-cm arm-band, found an average variation of about 10 mm., giving their average as 46 mm. in adults as compared with 35.7 mm. in children. This is in close accord with the at present accepted belief that the pulse pressure is equal to about one-third the systolic pressure.

The same factors which influence blood-pressure from time to time in adults act with even greater force upon blood-pressure in children.

It is generally believed that all things being equal the weight and size of the child, rather than the exact age, is the important factor in determining the actual systolic pressure level.

Orthostatic Albuminuria in Children.—The occurrence of this condition and its relation to and effect upon blood-pressure is of considerable interest and importance to the

¹ *Brit. Med. Jour.*, Apr. 17, 1915, 1, 2833.

pediatrician, as here the blood-pressure test will serve to differentiate this type of urinary disturbance from those of more serious import. In this connection the occurrence of this type of albuminuria is rather interesting. Thus K. Bugge¹ examined over a thousand school children and found albumin present in 14.9 per cent. in which it had no relation to cardiovascular or renal conditions. In demonstrating the effect of physical exercise upon this condition Bugge was able to demonstrate the development of transient albuminuria in 20 per cent. of healthy boys after gymnastic exercise.

This type of albuminuria is sometimes seen in convalescents and as an accompaniment of cardiovascular weakness in the presence of low blood-pressures, and is here probably a reflection of an abnormal reaction of the vasomotor relations of the kidneys.

Bass and Wessler's² observations show that the blood-pressure of children suffering from orthostatic albuminuria differs but little from that of normal children, in spite of an apparent vasomotor insufficiency, which many of these children show.

Blood-pressure reactions, both in the upright and in the recumbent postures and after exercise, revealed no characteristic anomaly. Nor were they able in any way to correlate the blood-pressure findings with the findings in regard to the size and strength of the heart or the pulse rate. They do not believe that children with orthostatic albuminuria, accompanied by cardiovascular symptoms, can be differentiated by means of blood-pressure tests.

¹ *Norsk. Magazin. f. Lægevid.*, December, 1914, lxxiv, 12.

² H. H. Bass and H. Wessler, *Arch. Int. Med.*, January, 1914, xii, 1.

Pneumonia.—The fact that the symptoms of death of children from pneumonia are largely those of vasomotor failure, makes the study of this reaction of particular importance. Howard and Hoobler¹ have studied the effect of fresh air on patients with acute pneumonia. They find that there is always a rise in blood-pressure following removal from a warm well-ventilated ward to the cold balconies of Bellevue Hospital. This rise is apparent at the expiration of a half hour or more, after removal, and reaches its maximum only after about two hours, remaining constant thereafter, even for thirty hours, and there was noted no tendency for the pressure to fall as if from exhaustion by the effort. Upon return to the warm well-ventilated ward a fall is apparent in from fifteen to twenty minutes, usually reaching its lowest point in an hour, when it remains unless influenced by the course of the disease, by stimulation or by a return to out-of-doors. They concluded that this rise of 10 or 15 mm. of mercury was due to the effect of the cold air upon the skin of the face and on the nasal mucous membrane, and not to the purity of the air.

Acute Nephritis.—Lenox Gordon reports nine cases of acute nephritis in all of which the blood-pressure was found to be above normal. In some cases this elevation is marked, becoming of distinct diagnostic value, as in no other disease of childhood is there to be found such a marked rise in pressure. As in adults, the pulse pressure has a tendency to be increased, owing to defective elimination and the consequent retention of toxic substances

¹ J. Howard and B. R. Hoobler, *Am. Jour. Dis. of Children*, May, 1912, iii, 5.

which directly affect the elasticity of the arterial walls and the permeability of the capillary system.

The frequent occurrence of general edema in this disease calls attention to the importance of the element of error introduced into pressure readings made through tissues where edema exists.

The Sphygmomanometer in Asphyxia Neonatorum.—

Ballard¹ speaks highly of the value of the sphygmomanometer as a means of detecting the persistence of the fetal heart beats in babies born in asphyxia. He cites four cases, in which neither the radial pulse nor the heart beat could be detected, and yet in which the apparatus demonstrated slow brachial pulsations.

¹ P. Ballard, *Presse Médicale*, 1913, Mar. 22, No. 26.

CHAPTER XII

ACUTE INFECTIONS

In the study of infectious diseases, the routine use of the blood-pressure test offers an almost unlimited field of usefulness, which in the light of present knowledge, no physician can afford to neglect. Naturally this test offers little in the way of diagnosis, but for prognosis and as a guide to treatment it becomes a constant and a most reliable aid.

TOXEMIA AND BLOOD-PRESSURE

As a general rule, in acute infections in a robust individual, where the onset is sudden, there is noted an early rise in systolic pressure amounting to not more than 10 or 15 mm. Hg. During the course of the invasion, as the toxemia develops, there is a gradual fall in the systolic pressure level, in which the diastolic participates, usually to less extent, the result being a narrowing of the pulse pressure. As convalescence advances there is a gradual return to normal values. The degree of depression and the rapidity of the return of the convalescent's pressure to normal will be influenced by (a) the duration of the disease, (b) the profoundness of the toxemia, and (c) vitality and resistance of the patient. The depression is both marked and prolonged in typhoid fever and in pneumonia.

Complications may alter the picture in several ways. Thus, in convalescence from prolonged fevers, when the

patient assumes the erect posture, the systolic pressure may be found to fall, while the diastolic may remain unchanged. This reduction in pulse pressure, tends to cerebral anemia which reflexly increases heart rate, thereby causing unnecessary cardiac strain. This probably explains the cardiac disturbances seen so often in convalescents from acute infections and which should be guarded against by frequent blood-pressure observations, permitting a gradual return to physical activity only when indicated by the findings. Even late in the convalescence or after apparent return to normal, physical exertion may cause the same disturbance, from which the heart may not recover for weeks or even months. So frequently is this condition encountered that R. N. Willson¹ has said that "we may state that there occurs probably no instance of perfect cardiac recovery from an acute micro-organismal invasion."

The incidence of renal irritation or of an acute nephritis during the course of an acute infection will speedily be accompanied by a sharp rise in systolic pressure even before urinary or other signs appear.

Violent purging and diarrhea will accentuate the usual fall seen during the progressive stages of acute infections.²

Venous pressure tends to fall coincidently with the systolic during the active febrile stage, unless the heart becomes embarrassed, when the venous pressure rises and adds an additional burden to the already laboring heart.

In acute infections the basis for application of the test is the experimental evidence of the influence of bacterial

¹ *Penn. Med. Jour.*, December, 1914, Vol. iii, No. 12.

² Nelson and Hyland, *Jour. A. M. A.*, Feb. 8, 1913, lx, 6, p. 436.

poisons and toxemias on the vasomotor system. Sajous¹ has brought forward a theory of the relation of the adrenal gland to the dangerously low blood-pressure found in the terminal stages of acute infections, especially in pneumonia and typhoid fever. Sajous quotes Goldzicher who reaches the conclusion that in septicemia the appearance of low blood-pressure is to be ascribed to insufficiency of the adrenals. This relation, if found to be the true explanation, when generally recognized may yield a rich harvest of recoveries.

Methods of Study.—In the study of infectious diseases, single observations are valueless because of the lack of normal figures for comparison. Careful daily observations should be made and recorded and if the pressure tends toward a dangerous hypotension, the periods of observation should be shortened to meet the requirement.

PNEUMONIA

The danger in pneumonia is largely cardiac. The blood-pressure is usually low. The toxic influence upon the vasomotor mechanism and upon the heart muscle may be such that this organ, in its weakened state, is unable to pump strongly enough to maintain an efficient arterial pressure, in the face of vasomotor relaxation. Unfortunately, we are not able to depend entirely upon the blood-pressure test as a guide to prognosis in pneumonia. Statistics are not in accord. It also appears that death occurs sometimes in cases showing higher pressure, while some recover with very low pressure, so that a low systolic pressure does not necessarily always have fatal import.

¹ *Monthly Cyclo. Med.*, December, 1911.

Other factors enter here, particularly the previous history and personal habits of the case, especially addiction to alcohol.¹

Gibson's Rule.—So called because first formulated by Gibson² in 1908. This blood-pressure pulse ratio has been

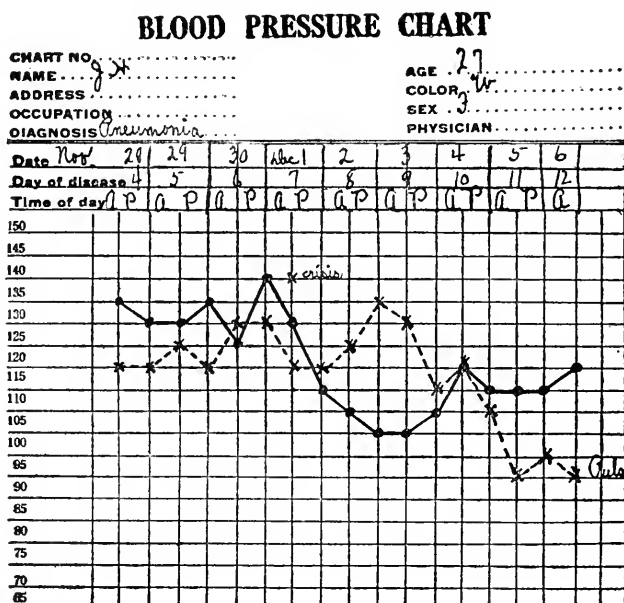


FIG. 37.—Showing close relation between pulse rate and blood-pressure and application of Gibson's rule. Symptoms of collapse developed after crisis and continued until normal relation was reëstablished during convalescence.

the subject of much controversy and an occasional frank attack Newburgh and Minot,³ nevertheless the bulk of clinicians adhere to it and find value in its application (see Fig. 37). This so-called rule as originally stated by Gibson

¹ L. H. Newburgh and G. R. Minot, Arch. Int. Med., July, 1914, xiv, No. 1.

² Edinburg Med. Jour., January, 1908.

³ Loc. cit.

is as follows: "When arterial (systolic) pressure expressed in millimeters of mercury does not fall below the pulse rate expressed in beats per minute, the fact may be taken as of excellent augury while the converse is equally true." No one should accept this statement unconditionally, for there are many exceptions. In applying the rule the physician will avoid being led into the error of relying entirely on it, by giving due weight to such other factors, as the violence of infection, an alcoholic history or not, the presence of chronic disease, etc. Also the mere disturbance of this ratio for a short period is of little significance, becoming important only when the ratio persists for some time in the face of stimulating measures.

Concerning the practical value of this test, G. A. Gordon¹ states that in a recent series of cases studied by him there were no fatalities among those whose quotient remained B.P. > P.R.

Alex. Lambert² studied forty-eight cases; of these twenty failed to maintain B.P. > P.R. and of them six or 42 per cent. died, while the twenty-eight in whom B.P. > P.R., five or 17.09 per cent. died. Gundrum and Johnson³ report observations on thirty institutional cases between the ages of eighteen and sixty, twenty of whom were alcoholic. They had nine deaths (mortality 30 per cent.) among those whose systolic blood-pressure on admission ranged between 65 and 122 mm. Hg. with a pulse of 68 to 148.

Eighteen cases maintained B.P. > P.R. and seventeen

¹ *Edinburgh Med. Jour.*, January, 1910.

² *Jour. A. M. A.*, Dec. 2, 1911, lvii, 23, p. 1827.

³ F. F. Gundrum and E. E. Johnson, *Calif. State Jour. of Med.*, May, 1912, x, 5.

or 94 per cent. recovered while of the twelve who showed B.P.<P.R., nine or 75 per cent. died. These observers feel that they are greatly aided in treatment by following the rise and fall of pulse pressure and systolic pressure.

Auscultatory Sounds as Aids to Prognosis.—The second and third phases when good and clear are considered favorable signs in pneumonia as they indicate heart strength (see page 99). The second phase is the one most quickly lost, from heart weakening in pneumonia, and its presence throughout the disease is therefore a good sign.

The muffling or weakening of any of the sounds should be looked upon with suspicion as such changes indicate a relaxation of peripheral resistance and a failing heart; the addition of arrhythmia to these variations is a further indication of the same adverse change.¹

Newburgh and Minot noted among other findings that the blood-pressure curve in pneumonia did not suggest a failure of the vasomotor center, which conclusion has been amply confirmed experimentally by W. T. Porter. Both were able to demonstrate that it was not the vasomotor center but the myocardium itself that failed in fatal pneumonia. Even more convincing are the microscopic findings of Willson who in every instance was able to detect myocardial changes due either to a toxemia or to a local infection.

F. Tice² found Gibson's ratio to hold good in 88 per cent. of cases and considers it of value not only in prognosis but also of much assistance in treatment, where it is a valuable guide to the administration of stimulants.

¹ A. A. Howell, *Jour. A. M. A.*, lxii, 16, Apr. 18, 1914.

² *Jour. A. M. A.*, Mar. 27, 1915, lxiv, 13.

Normal Pressure Level in Pneumonia.—G. M. Piersol¹ followed ten cases, three of whom ran an average pressure above normal throughout the disease, four maintained a pressure that was normal for the age of the individual, three fell below during the active stage but quickly rose after the crisis to an approximately normal level.

Relation of Pressure to Crisis.—Piersol has seen a rise, in a woman aged fifty, amounting to 40 mm. systolic and 30 diastolic in less than twenty-four hours after crisis.

In mild cases there is not much change at the crisis, especially in young adults, although pressure will usually be lower after crisis and during convalescence than during the height of the disease. A. Lambert² has not noted any sudden fall at crisis, but a gradual return to normal in the cases that recover, the pulse usually diminishing in frequency before the blood-pressure rises, though occasionally the reverse may be true.

In cases where neither pulse nor fever is high, but the blood-pressure very low, below 100, with $BP > PR$ the cases recover, but in these cases the pressure is very slow in rising and these hearts should be carefully watched and guarded.

In cases with chronic nephritis, though the pressure may remain between 170 and 220 yet the patients may die.

The reading of blood-pressure in arteriosclerosis and nephritis does not give a clue to the cardiac conditions and in these cases Gibson's ratio does not hold. Dyspnea may cause rise in blood-pressure, the pressure level is also influenced by the day of the disease.

¹ *Loc. cit.*

² *Jour. A. M. A.*, Dec. 2, 1911, lvii, 23, p. 1829.

TYPHOID FEVER

All authorities agree that typhoid fever is a disease of hypotension, and that the depression in systolic pressure appears early, and is progressive (unless interrupted by complications) throughout the active progress of the disease; further, the depression of pressure persists for a relatively long time, even after the temperature has returned to normal and the patient is well established in convalescence. The preëxistence of cardiovascular or renal lesions will modify the pressure readings and when variations from the usual course are met such complications should be suspected. Even in these cases a hypotension (but with readings above the patient's normal age level) may be demonstrated, if we were able to obtain a knowledge of the patient's pressure before the onset of the disease.

Vasomotor weakness of the splanchnics caused by the toxins of the disease is usually considered to be the cause of the pressure depression.

The pressure during the first week, when cases are rarely seen by the physician, may not be sufficiently modified to be noticeable.

Blood-pressure Readings.—The systolic pressure in typhoid fever, after the first week in young adults, will usually register below 100, progressively falling until the fourth week, when it usually begins to ascend.¹ Pressures of 85 and 90 systolic are not uncommon, while a systolic pressure of 74 mm. has been recorded (Crile). The author as a matter of convenience, and as an aid to comparison, considers that the pressure in typhoid fever averages about

¹ Geo. W. Crile, *Jour. A. M. A.*, xl, 1903.

110 mm. Hg., during the first week, dropping about five points per week during the next three, and then begins to return to normal as convalescence is established.

The Diastolic Pressure.—As in any case of pressure variation, the diastolic pressure tends to follow the systolic, though the movement is usually less. This results in a reduction in pulse pressure, which is present during the active stages of the disease and which becomes gradually lost as the case recovers, to return temporarily if the heart is overstrained.

The blood-pressure changes in typhoid fever are so characteristic that in obscure cases of continued fever, a carefully prepared blood-pressure chart may aid in clearing up the diagnosis. Blood-pressure readings should be made as regularly and frequently as are the pulse and temperature observations, because here, as in any other conditions when comparatively rapid alterations are to be expected, an occasional reading is valueless. This fact is demonstrated by the evidence afforded by systematic blood-pressure records which may call attention to the occurrence of such complications as perforation or hemorrhage. They may also assist in an early diagnosis, or in demonstrating the effect of therapeutic measures, particularly baths, upon the cardiovascular apparatus.

Complications.—*Perforation.*—Crile¹ and Cook and also Briggs² note that in typhoid fever with perforation and peritonitis, there is an early and decided rise, which is followed by a fall as toxemia increases. This was found to be the invariable rule by Crile in twenty surgical patients.

¹ *Jour. A. M. A.*, May 9, 1905.

² *Johns Hopkins Hos. Rep.*, Vol. xi, 1903.

A sudden rise in pressure in the course of a case of typhoid is almost positive evidence of a perforation, though we should remember that a stationary pressure is no indication that the catastrophe has not occurred.¹

Hemorrhage.—There is a rapid fall in blood-pressure without the initial rise, by which fact it may be separated from the preceding. The degree and rapidity of the fall in some measure indicates the extent of the hemorrhage. The pressure tends, upon the arrest of hemorrhage, to return rapidly to almost the level noted before the hemorrhage occurred.

Value in Treatment.—In the treatment of the disease, a study of blood-pressure will be found to be of great value. It shows the best mode of combating the circulatory failure and indicates whether our efforts should be directed toward improving peripheral resistance, or toward heart stimulation, and gives a clear indication whether the case is receiving too little or too much fluid for the capacity of the circulation.

Nowhere is the effect of stimulatory measures shown to greater advantage than by the changes in blood-pressure relations shown by the sphygmomanometer through records taken before and after a bath.

Secondary and Late Effects of Typhoid Infection upon Blood-pressure.—Acute infections are now recognized as among the most frequent as well as the most insidious causes of arteriosclerosis and it has been shown that typhoid fever is particularly involved in the early production of arteriosclerosis (see page 257).

¹ A. L. Sheppard, *Lancet*, May 11, 1907.

DIPHTHERIA

The effect of the diphtheria toxin upon muscular tissue throughout the body, and upon the heart muscle in particular, has long been a grave concern of the practising physician, heart death after diphtheria being an all too frequent sequela. The routine estimation of blood-pressure therefore becomes an important prognostic measure, particularly in this disease.

As in other infections, the blood-pressure tends toward subnormal during invasion, with a gradual return toward normal during convalescence.

From a clinical study of 179 cases of diphtheria Rolleston¹ found a subnormal pressure in sixty-three cases or 35 per cent., the extent and duration bearing a direct relation to the severity of the faucial attack. The highest readings were found during the first and the lowest during the second week. The normal tension was usually reëstablished by the seventh week. Evidence of dyspnea (partial asphyxia) in laryngeal cases caused an elevation in pressure. Tracheotomy in these cases was followed by an immediate fall of 20 to 40 mm. The effect of serum administration was a rise in pressure in 40 per cent. of cases. Albuminuria did not cause a rise in pressure, except in one case with uremia.

In studying the relation of blood-pressure in diphtheria to myocardial alterations Bruchner² examined critically 200 cases of this disease. He found that mild cardiac involvement did not affect the normal blood-pressure curve, and that cases with irregular blood-pressure showed

¹ J. D. Rolleston, *Brit. Jour. of Children's Diseases*, October, 1911.

² *Deutsche med. Wochen.*, Oct. 28, 1909.

various clinical pictures. Every case of marked fall in pressure was associated with definite signs of myocarditis. Falls amounting to as much as 50 mm. (Gärtner's tonometer) appeared only with severe myocarditis. This was the greatest drop in which recovery occurred. A steady progressive fall in pressure was present in the fatal cases. In every case, with one exception, marked falls in pressure were accompanied simultaneously by signs of cardiac involvement; in one case only did the fall precede the clinical signs.

Anaphylactic Shock.—This fortunately rare, although usually fatal, condition is believed to be due to a toxic constriction of the bronchial tree which results in a condition of strangulation. As far as known we believe that this process is primarily accompanied by a rise in pressure.¹ This rise however, is, so transient that it is rarely observed; so that in the average case, if pressure readings are made, there will usually be found a marked hypotension, which is the usual accompaniment of acute cerebral anemia (see page 379).

SCARLET FEVER

In the study of the systolic pressure in a series of cases of scarlet fever, Rolleston² noted that the extent and duration of the depression was usually in direct relation to the severity of the initial attack, and that while the greatest number of normal readings occurred during the first week, there was nevertheless a predominance of lowest readings in the same week. The great majority of low readings

¹ Auer and Lewis, *Jour. A. M. A.*, 1909, Vol. liii.

² J. D. Rolleston, *Brit. Med. Jour. of Children's Diseases*, October, 1912 lx, p. 444, 106.

occurred in the second week, while normal tension was usually reëstablished some time during the fourth week. In the majority of cases the blood-pressure was lower during convalescence than in the active stage, although nothing of a characteristic nature regarding the relative height of the pressure during the acute stage as compared with the nature of the convalescence has been found.

With the exception of nephritis, the ordinary complications have little if any effect on blood-pressure. The chief value of the sphygmomanometer in scarlet fever is in detecting the occurrence of marked renal irritation as, with the development of an acute nephritis, there is always a sudden and marked rise in arterial pressure. Buttermann¹ has observed a rise of more than 50 mm. within twenty-four hours after such an occurrence.

The presence of marked hypotension, especially if accompanied by other signs of vasomotor insufficiency, calls for appropriate measures directed toward circulatory support.

**Rolleston's Table Showing the Number of Cases in Each Week
in which the Highest Readings were Recorded**

	First week	Second week	Third week	Fourth week	Fifth week	Sixth week
Severe.....	11	1	0	1	0	1
Moderate.....	17	1	4	2	2	0
Mild.....	39	4	6	3	0	0
	67	6	10	6	2	1

An unusual range of systolic blood-pressure readings may be met in scarlet fever in children; thus Rolleston

¹ *Arch. f. klin. Med.*, lxxiv, p. 11.

has noted a pressure of 70 in a girl aged six in a mild attack, and a pressure of 150 in a girl aged fifteen, where there were no signs of nephritic involvement.

The average case will run between 105 and 75, the individual readings being modified as in the normal, particularly by the age factor. The occurrence of laryngitis, owing to the respiratory obstruction, may be expected to cause a rise of from 20 to 30 mm.

CHOLERA

Hypotension is the rule. Low blood-pressure during the stage of collapse is a valuable guide to the necessity of transfusion. The blood-pressure is always below 100. The most satisfactory treatment, or the one most likely to combat complications, such as uremia, is administering the intravenous solution of adrenalin. By this means in one epidemic the death rate was reduced almost one-half.¹

In cases showing very low pressures, Rodger and Megraw² consider the estimation of blood-pressure of great value in determining the frequency and quantity of saline solution to be injected; they state that the restoration of the circulation thus brought about, not only affords great relief to the patient but materially aids in the elimination of toxins through the kidneys and the bowel.

MALARIA

In chronic malaria, in the absence of complications, the blood-pressure, as would naturally be supposed, is usually low. In acute cases the pressure curve follows the rise and fall of the fever, being high during the chill and in the

¹ Leonard Rogers, *Therapeutic Gazette*, Nov. 15, 1909.

² *Indian Med. Gaz.*, March, 1908.

febrile stage, falling as the sweating process begins, and remaining low until the onset of the next rigor. As this phenomenon is the natural accompaniment of the conditions with which they are associated, irrespective of the cause, the findings by the sphygmomanometer have but little significance in this disease.

EPIDEMIC CEREBROSPINAL MENINGITIS

G. C. Robinson¹ found that elevated intracranial pressure is almost a constant phenomenon in epidemic cerebrospinal meningitis, and that this usually is accompanied by elevated blood-pressure. This rise in the blood-pressure appears to bear some relation to the severity of the disease, being high when the symptoms are severe and low when mild, and during convalescence. Robinson's observations in reference to the effect of lumbar puncture are not in accord with those of later observers, particularly Sophian.² This author states that the withdrawal of fluid in meningitis is usually accompanied by a drop in blood-pressure, sometimes quite a marked one, especially if the amount of fluid withdrawn be great. He depends absolutely upon this observation as a guide to the amount of fluid to be withdrawn and the quantity of serum to be injected in the treatment of this disease. Sophian's procedure is as follows: Commencing with an ordinary case with a systolic pressure of 110 mm. Hg., fluid is withdrawn until a fall of pressure amounting to 10 mm. in adults and 5 in children occurs. If the pressure begins to fall quickly, the rapidity of the flow should be reduced. Occasionally

¹ *Arch. Int. Med.*, May, 1910, Vol. B.

² Abram Sophian, *Jour. A. M. A.*, Mar. 23, 1912.

there is no fall in pressure (there may even be a rise) and the indication here is to remove as much fluid as possible or until the cerebrospinal pressure is normal. After the fluid has been withdrawn the serum is warmed and then slowly injected. When, contrary to expectations, the blood-pressure continues to fall, the indication here is as follows: Beginning with a 10-mm. drop from fluid removal, the injection is terminated with the development of a total drop of 20 mm. By this method the average dose of serum is smaller, averaging not more than 25 c.c. in adults and in children in proportion.

Muscular movements and pain may interfere with the correct observation of pressure while the presence of internal hydrocephalus usually causes an increased systolic pressure.

OTHER ACUTE INFECTIONS

In the other acute infectious diseases there is little to state that is of practical importance regarding the blood-pressure, because many of them are so mild as to have no appreciable effect upon arterial tension, and also because observations as far as they have been made, shed very little light. In general it may be stated that the development of toxemia from any cause, results in depression of the normal pressure curve which tends to return to normal with relief from the toxemia.

CHAPTER XIII

CHRONIC INFECTIONS

TUBERCULOSIS

Statistics upon blood-pressure observations in tuberculosis made by different observers show wide variations in the results recorded, though they agree upon two points: first, that the systolic blood-pressure is lessened when this disease is active and that it becomes progressively lower as the disease advances, and second, that it rises toward normal as the process is arrested and the disease cured. The first of these points is exemplified by the following statistics which have been collected, tabulated and averaged by Pottenger.¹ These observations are grouped for comparison according to recognized divisions into first, second and third stages, and are as follows:

In Strandgaard's series² the systolic pressure averaged in the

First stage.....	125 mm. Hg.
Second stage.....	121 mm. Hg.
Third stage.....	118 mm. Hg.

Burckardt,³ making his examinations at Basel, found that the average systolic pressure in the

First stage.....	107.6 mm. Hg.
Second stage.....	104.6 mm. Hg.
Third stage.....	100.3 mm. Hg.

¹ *N. Y. Med. Jour.*, Aug. 31, 1912, p. 418.

² N. J. Strandgaard, *Internat. Zentrallb. f. Tuberkulose*, 1908, p. 224.

³ *Zeitsch. f. Tuberkulose*, v, p. 19, viii, pp. 465-8.

Ingersheim¹ found in the

First stage.....	100.4 mm. Hg.
Second stage.....	97.3 mm. Hg.
Third stage.....	95.4 mm. Hg.

Pottenger² examined twenty normal persons and compared the findings with those in 135 tuberculous patients divided according to stages of the disease. The average systolic and diastolic pressures were as follows:

	Systolic	Diastolic
20 normal individuals.....	120	108 mm. Hg.
11 patients in first stage.....	106	78 mm. Hg.
21 patients in second stage.....	108	81 mm. Hg.
103 patients in third stage.....	103	75 mm. Hg.

Thus we have average blood-pressure readings as follows:

First stage.....	from 100.4 to 125 mm. Hg.
Second stage.....	from 97.3 to 121 mm. Hg.
Third stage.....	from 75.4 to 118 mm. Hg.

That a depression in systolic level is an early sign of tuberculosis is accepted by most observers. The importance of this point has been emphasized by Cook who makes the following significant statement: "When low blood-pressure is persistently found in individuals or in families it should put us on our guard for tuberculosis," and Haven Emerson,³ dwells upon the fact that hypotension should be sought for just as carefully in a physical examination as we customarily search for pulmonary signs. Many cases of so-called idiopathic low pressure will be found later to develop signs of pulmonary involvement so that hypotension, when otherwise unexplained, should suggest a

¹*Zeitsch. f. Tuberkulose*, viii, p. 467.

²*Arch. Int. Med.*, October, 1909.

³*Arch. Int. Med.*, April, 1911.

careful examination for tuberculosis, particularly of the lungs.

N. J. Strandgaard¹ believes, with others, that a low pressure may help to differentiate tuberculosis in doubtful cases,

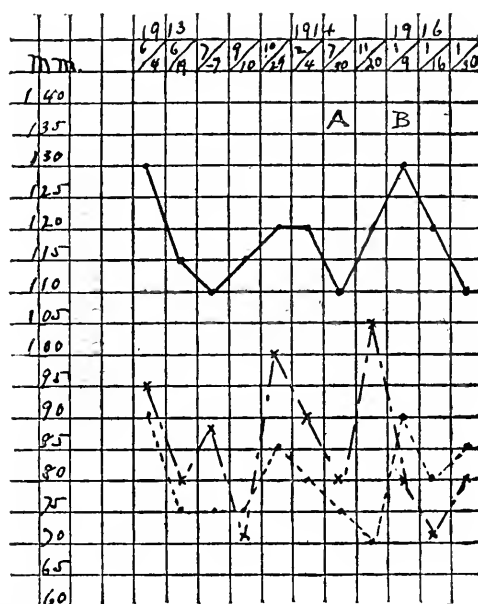


FIG. 38.—Male. Married, aged thirty-seven. Chronic phthisis. Has already had several hemorrhages, there is a small cavity in right upper lobe. Periods of depression of systolic occurred coincidently with exacerbations of the disease. The observation at (A) was made after the patient had suffered a severe attack of grippe.

Between periods A and B condition has improved in spite of an attack of grippe and weight increased 9 lb. Tuberculin treatment begun(B). Last examination; weight stationary, and temperature, pulse and respiration normal, no cough, appetite and stomach in good condition.

and that it will show whether a tubercular process is active or latent, for practically all authorities have agreed that hypotension is present in tuberculosis, that it progress-

¹ *Hospitalstidende*, Oct. 16, 1907, l, 42, p. 1113.

ively increases with the extension of the process and that recovery from the hypotension indicates the arrest of or improvement in the pulmonary lesions (see Fig. 38). Return to normal pressure is commonly found in those who are cured, as a continued low level appears never to exist in the presence of pulmonary improvement.

In spite of the fact that a lowered systolic pressure is practically always the accompaniment of an active pulmonary tuberculosis, it is believed that the degree of involvement does not necessarily bear any relation to the systolic level, so that observations do not throw any special light in this direction.¹

This variation in systolic depression in active cases has been ingeniously explained by Pottenger² who states that we have several modifying factors which are opposed to the generally depressive effect of the toxemia on the vascular tonus and on the vasomotor centers. Thus, early in the disease there is an increased number of heart beats which results in an hypertrophy of the right ventricle; followed later, when pulmonary pressure is increased, by an augmentation of strength of the right ventricle, a condition which is maintained as long as the muscle is able to meet the additional strain. Furthermore, the toxins of the disease, which continue to circulate, directly affect the arterial wall and bring about a condition of generalized arterial fibrosis, so that the longer the disease exists the greater the development of this condition. The relation of arteriosclerosis to tuberculosis is conclusively shown in

¹L. S. Peters, *Arch. Int. Med.*, August, 1908.

²*Loc. cit.*

the study of 162 patients where the following results were obtained:

Condition of radials	Duration of the disease		
	Less than one year	One to two years	More than two years
Palpable.....	14	20	60
Non-palpable.....	14	21	33

The effect of altitude upon systolic pressure in the tuberculous has been the subject of investigation by a number of observers, and the consensus of opinion is generally that at an altitude of 6000 ft.¹ the blood-pressure is increased as compared with the pressure in tubercular cases seen at sea level. This opinion is not accepted by F. C. Smith² who, after a careful series of observations upon groups of both normal and tuberculous individuals, found the effect of altitude on the systolic blood-pressure in the tuberculous to be insignificant. He believes that the effect of the change has been overestimated, and that when due allowance had been made for disturbing factors, there was nothing observed by him to show that an altitude of 6230 ft. has any pronounced influence upon this phenomenon.

Prognosis.—From a prognostic standpoint blood-pressure findings are of great value in tuberculosis for, although there is no relation between the degree of involvement and the systolic level, there is a constant relation between the degree of toxemia and the blood-pressure. Emerson³ believes that we can place as much dependence upon altera-

¹ Peters and Bullock, *Arch. Int. Med.*, October, 1913, xii, No. 4.

² *Jour. A. M. A.*, May 29, 1915, lxiv, 22.

³ *Loc. cit.*

tions in blood-pressure as we now do upon studies of the pulse and temperature. It must not be forgotten, in this connection, that a study of the diastolic pressure is of equal importance, and that both in tuberculous suspects, and during the course of the disease, changes in pulse pressure are most significant, not only as indicating the degree of the toxemia but also as showing the effect of therapeutic measures upon the cardiovascular system.

In order to determine the relation of blood-pressure to prognosis Strandgaard¹ studied the pressures in 174 adults and 163 children who were cured or materially improved. These showed an average systolic pressure of 125 mm. Hg, as compared with 234 patients who were moderately or not at all improved who showed an average of 121, while in twenty-one adults and thirteen children in whom the disease followed a progressive course, seventeen of whom succumbed, the average was 108 mm. Hg. In each group there was a wide range of blood-pressure in individual cases, but the general average of 125 mm. Hg. in the first and 108 mm. Hg. in the last group are very significant.

Theory of Cause of Reduced Pressure.—It is probable that in tuberculosis as in other infections, the systolic depression is due to the effect of the circulating toxins of the disease affecting the arterial coats, cardiac nutrition and the vasomotor centers. It has also been suggested that the adrenals are in close relation to the hypotension, although Sézary² investigating this possible factor, was unable to find any relation between the state of the supra-

¹ 1907 reference.

² *Arch. des maladies du coeur*, February, 1910, iii, 2.

renal glands and the low pressure found in tuberculosis, and in proof cites a number of cases, examined at autopsy, where there was almost complete destruction of these glands and yet which during life showed a relatively high blood-pressure.

Hemorrhage.—During the earlier studies of the systolic blood-pressure in tuberculosis, most authorities call attention to a period of increased pressure preceding or accompanying hemoptysis in patients previously showing hypotension. Recently, however, Smith¹ carefully studied the systolic readings in a series of cases having pulmonary hemorrhage, as compared with an equal number of cases who were free from this complication, from which he concludes that there is nothing found to support the contention that hemoptysis is more likely to occur in those with a high systolic pressure.

Accidental Complications.—It is generally believed that the intercurrent development of an arthritis or of diabetes may alter the blood-pressure picture by causing a normal or an elevated systolic level to develop. It should also be borne in mind that the presence of an old nephritis will affect the readings and will usually cause the systolic pressure to be above normal, although the elevation of pressure does not reach the level that would ordinarily be expected from the degree of kidney involvement. The incidence of intercurrent acute infections has no effect upon the systolic pressure, neither has the administration of alcohol in normal medicinal amounts. Hyperalimentation is considered by Strandgaard to be a cause for a rise in systolic pressure, and for this reason he urges often re-

¹ *Loc. cit.*

peated determinations of blood-pressure as a guide for the proper forced feeding of tuberculous cases. The usual effect of a change from warm to cold air is to produce a rise in systolic pressure and Hoobler¹ has found that the more advanced the disease, in cases treated within doors, the lower the pressure will be and that a demonstrable increase in systolic pressure occurs with transfer to the open air.

Effect of Exercise on the Tuberculous.—A valuable and most complete study of the effect of exercise upon tuberculous patients has been made by L. S. Peters and E. S. Bullock.² A definite plan was outlined and careful study and accurate records made. Six men were used. Three were excellent cases both pulmonarily and physically, two fairly arrested, far advanced cases; and one a new recruit, with normal temperature, but poor physical condition. The points in this report are so well taken and the table shows so graphically the results obtained, that they are copied here in full.

“All six were started with a fifteen minutes’ walk the first day. The pressures on starting of the three able-bodied men were 138, 132 and 148 respectively. On their return the pressures were 138, 144 and 153. After an hour’s rest the readings were 138, 142 and 158, showing that apparently the exercise was not harmful. The two fairly well arrested, far advanced cases, started out with pressures of 164 and 124, returning with 146 and 130, and after resting 164 and 118. The first man was not accustomed to exercise in any form, as is clearly shown in a drop of 18 mm.

¹ *Amer. Jour. Dis. of Children*, 1912, iv.

² *Med. Rec.*, Sept. 14, 1912, p. 463.

Hg., with a return to the original after an hour's rest. The over-exertion in the second man is evident, for we find after resting that there is a drop of 6 mm. from the original reading recorded after the return from exercise."

"The new recruit started with 146, returned with 138 and after rest his reading was 127. The over-exertion in this instance is well illustrated by the pressure findings and was further substantiated by the marked fatigue, breathlessness, and rapid heart action of the individual. This experiment was carried on for a period of six days, each day's exercise being graded by the previous day's results in blood-pressure. The table of these findings, which we here append, shows at a glance that we are able to control the readings by an increase, a decrease, or a repetition of the exercise. Whenever a man showed a drop of 6 or more mm. Hg. after rest or a marked drop on returning, even though this disappeared after resting, we decreased the exercise. If there was a slight drop after returning we repeated the same exercise the following day or until we maintained an even standard, when the walk was increased. It is interesting to note that in one of the three *excellent cases* the pressure remained practically the same even with a walk of one and a half hours, and later this same man took walks of two hours in the morning and two in the afternoon with no change in pressure and no evil results. The other two after a few repetitions were able to do the same."

Improvement in subjective symptoms follows the effect of blood-pressure elevation, and persists if the pressure can be maintained at a higher level than that existing before such treatment.

Case No.	Blood-pressure			Pulse			Remarks
	Before	After	After rest	Before	After	After rest	
1	138	138	138	1st day. 15 minute walk.
	138	141	150	92	98	80	2d day. Increased to 30 minutes.
	140	139	150	98	92	76	3d day. Increased to 40 minutes.
	142	140	140	96	106	80	4th day. Increased to 1 hour.
	140	142	138	88	88	76	5th day. Increased to 1 hour, 15 minutes.
	142	138	140	88	88	76	6th day. Increased to 1 hour, 30 minutes.
2	132	144	142	1st day. 15 minute walk.
	126	122	127	74	80	2d day. Increased to 30 minutes.
	126	135	126	66	76	68	3d day. Increased to 45 minutes.
	132	134	116	78	80	84	4th day. Increased to 1 hour.
	126	130	126	70	80	72	5th day. Cut to 45 minutes.
	126	124	136	73	76	75	6th day. Repeated 1 hour walk.
3	146	138	127	1st day. 15 minute walk.
	142	124	132	120	120	100	2d day. Repeated 15 minute walk at slower pace.
	140	145	130	120	118	96	3d day. Cut to 10 minutes at slow pace.
	132	145	142	120	120	112	4th day. Repeated 10 minute walk.
	142	150	140	116	120	108	5th day. Repeated 10 minute walk.
	138	146	142	118	120	104	6th day. Increased to 15 minutes.
4	148	152	158	1st day. 15 minute walk.
	146	146	135	88	84	72	2d day. Increased to 30 minutes.
	145	142	146	80	80	72	3d day. Repeated 30 minute walk.
	150	140	142	80	100	72	4th day. Increased to 45 minutes.
	145	140	158	84	80	76	5th day. Increased to 1 hour.
	140	142	138	76	80	80	6th day. Increased to 1 hour, 30 minutes.

Case No.	Blood-pressure			Pulse			Remarks
	Before	After	After rest	Before	After	After rest	
5	164	146	164	This man unused to exercise.
	146	134	144	118	120	106	1st day. 15 minute walk.
	140	150	138	100	104	100	2d day. Cut to 10 minutes.
	146	146	138	90	98	110	3d day. 10 minute walk.
	144	144	152	110	100	100	4th day. Increased to 15 minutes.
	138	144	146	104	104	92	5th day. Repeated 15 minute walk.
6	124	130	118	6th day. Increased to 25 minutes.
	115	114	110	110	120	108	1st day. 15 minute walk.
	122	117	118	100	100	84	2d day. Repeated 15 minute walk.
	115	110	115	100	110	100	3d day. Increased to 20 minutes.
	122	110	112	88	110	96	4th day. Increased to 30 minutes.
	115	114	114	96	96	88	5th day. Increased to 45 minutes.
							6th day. Decreased to 30 minutes.

SYPHILIS

The syphilitic heart and blood-vessels show clinically absolutely nothing characteristic, nothing which might not be found in other diseases. Our suspicions will be aroused when the patient is below the age of senile arteriosclerosis, when the involvement is essentially aortic, and that an incompetence, when the symptoms supervene rather suddenly and are not accompanied by fever, thus excluding acute endocarditis from any other cause.¹

As a primary cause of arteriosclerosis, syphilis is too

¹ Geo. Richter, *Med. Rec.*, Vol. lxxxii, No. 14, Oct. 5, 1912.

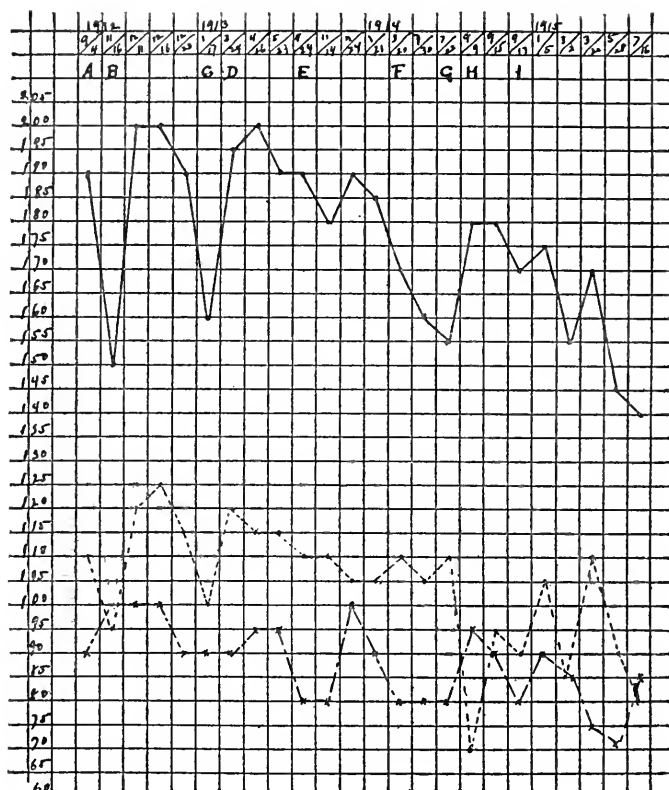


FIG. 39.—Male. Aged forty-nine. Specific disease, myocarditis and an old syphilitic and alcoholic hepatic cirrhosis.

Presented for examination on account of anasarca involving all extremities, with a large quantity of fluid in the abdomen, myocardium and pleuræ. Urine showed albumen very large amount ($3\frac{1}{2}$ Esbach), occasional casts and red blood cells. The heart sounds were extremely difficult to analyze on account of fluid. Dyspnea, cyanosis, extreme arrhythmia, etc., indicated grave heart involvement. Treatment combined specific, with infusion of digitalis and was most unsatisfactory on account of the patient's insisting upon traveling in spite of his condition. Between A and B patient's weight, due to loss of fluid fell from 273 to 250; and from B to C—from 250 to $216\frac{1}{2}$, during which the general clinical picture very much improved. At this time the treatment was changed to a modified Niemeyer pill, and later on account of intolerance, the mercury was reduced to a minimum. Between C and D conditions became very much worse and the weight returned to

well known to demand more than passing consideration here. In any history this disease should never be overlooked, but should be given due consideration as a predisposing cause in the production of those lesions of the cardiovascular and renal systems that are associated with hypertension (see Fig. 39). It is believed by many that a moderately high blood-pressure which can be traced to a previous syphilitic infection is more amenable to treatment and gives more satisfactory results than continued high pressure from other causes. This, however, remains to be proven. In the meantime, antisymphilitic treatment, particularly the use of the iodids, should be vigorously carried out.

During the early acute stages syphilis acts like any other general infection, in that it is usually accompanied by a moderate reduction in blood-pressure with some narrowing of the limits of pulse pressure. These changes are, as a general rule, so slight that they need not be considered in caring for the case; therefore, from a practical standpoint, the blood-pressure in acute syphilitic infection is of little value.

246. At *E* the addition of repeated purges to the digitalis treatment again reduced the weight to 220. At *F* the weight was 260 when active treatment was again instituted. At *G* the weight was reduced to 234, when there were no special complaints. Dyspnea not a factor, the color was fair, the pulse irregular in volume with a fair rhythm. At *H*, a severe infection of erysipelas of right leg followed a kick by a horse. Temperature 105°, probably contributing to the extreme pulse pressure record at this time.

At *I*, was convalescent and, due to continued rest, cardiac and circulatory conditions were excellent. Subsequent treatment has been entirely cardiac. The average systolic and pulse pressure indicates, as do also the patient's symptoms, that his condition is far better than when first seen.

CHAPTER XIV

BLOOD-PRESSURE IN METABOLIC, NEUROLOGIC AND MISCELLANEOUS CONDITIONS

The material contained in this chapter has been gleaned from a large volume of recent current literature. Its value to the physician will depend largely upon the method of its application in relation to the case in point, and will rather be corroborative and suggestive than distinctly diagnostic.

Addison's Disease.—Janeway has reported two cases of unquestioned Addison's disease, in which the systolic pressure tended downward. More recently Gibson¹ reports very low pressures in his series of cases. Two cases seen by the author showed a marked degree of hypotension. A. Rendel Short ("New Physiology") discusses the subject from the physiologic standpoint, and shows that, if the suprarenal veins are clamped for a few hours, thereby preventing the entrance of adrenalin secretion into the circulation, the blood-pressure rapidly falls. As the pathology of Addison's disease involves a degenerative process of the adrenal glands, we have the probable explanation of the low blood-pressures found in this disease. Improvement in subjective symptoms follows efforts directed toward blood-pressure elevation, and persist if the pressure can be maintained at a higher level than that existing before such treatment.

¹ *Brit. Med. Jour.*, Dec. 10, 1910.

Aviation Sickness.—In the *Medical Press and Circular* for August, 1911, reference is made to a communication by Crouchet and Moulinier to the French Academy of Sciences, in which they report their observations upon a number of aviators. They note two varieties of trouble resulting from flights in aeroplanes. The first is due to the altitude attained, and depends on differences in atmospheric pressure and temperature, and to changes in the chemical composition of the air found at high altitudes.

The second factor, which need not be discussed here, is the actual physical effort put forth. They consider the rapidity of ascent and of descent as most important, and recommend a reduction in the speed at which these changes should take place. The effect of ascent begins to be shown when a height of 1500 meters (4500 ft.) is reached, which causes quick, short respiration and tachycardia. There is usually a sensation of headache and moderate deafness. During descent there is a sensation of discomfort like that which accompanies a sudden descent in an elevator, violent palpitation and great noise in the ears. On landing the aviator is not free from the above sensations for a considerable time. Respiration quickly returns to normal but arterial hypotension which was found in most cases to be quite marked, persisted for a long time after the flight was finished.

Acute Alcoholism.—According to Raff¹ in a chronic alcoholic acutely inflamed with alcohol, there is a relative hypertension. During the first few days of treatment (withdrawal of alcohol) this hypertension persists and then

¹ *Deutsch. Arch f. klin. Med.*, 1913, cxii.

there is a gradual return to normal as the physical condition of the case improves.

Cardiac Asthma.—Attacks of sudden asthma occurring in the course of chronic cardiovascular and renal diseases are the direct result of some disturbance in the pulmonary circulation, probably due to a dilatation of the right ventricle and a rise in pressure in the pulmonary artery. This results in a condition of air hunger, in which defective alveolar interchange plays an important part. The resulting condition is virtually a partial asphyxia and is accompanied by a sharp rise in systolic pressure (see Fig. 40). Some authors even go so far as to state that this dyspnea is the underlying cause of hypertension although this is probably only the case in the essential or true bronchial asthma.

Pulmonary Edema.—It is believed that acute pulmonary edema is associated with a sharp rise in systolic pressure, although we have no knowledge of the condition of the circulation immediately preceding the actual filling up of the lung tissue so that we do not actually know whether the rise in pressure is primary or secondary to the pulmonary blocking and partial asphyxia due to the edema. Whatever this relation may be, it is self-evident that the usual case of cardiovascular disease, which has been interrupted by an acute pulmonary edema, is accompanied by a most striking pressure change which is almost as strikingly reduced, when the distressing symptoms are relieved (see Fig. 40).

Amblard¹ states that experimental research is amply confirmed by the clinical findings, and that in high pressure

¹ *Presse Medicale*, xix, 64.

cases further elevation in blood-pressure, due to cardiac insufficiency, may precipitate an attack of acute pulmonary edema; therefore careful attention to the blood-pressure, both systolic and diastolic, to determine an increase in pressure, or a functional failure of the heart, should direct attention to the need of immediate reduction in the maximal arterial pressure.

Cerebral Hemorrhage.—The rise in pressure accompanying the sudden development of a cerebral hemorrhage of traumatic or arterionephritic origin is caused by the local compression of a part of the brain. The effect of this sudden and local compression varies greatly according to the location of the compression, whether it be near or far from the fourth ventricle.

Pathology.—According to Cushing, it is not until the intracranial tension approximates the blood-pressure that profound changes in systolic tension occur. The effect of this altered relation is a stimulation of the vasomotor center, which is promptly followed by a rise in blood-pressure, sufficient to more or less reestablish the normal ratio between the intracranial and systolic blood-pressures. This change is a protective process, having for its purpose the maintenance of cerebral circulation, which is greatly interfered with and which rapidly results in death as the two pressures approximate, as equal pressure upon the two sides is incompatible with life. The ability of the vasomotor center to respond to this extraordinary demand will depend partly upon the rapidity with which this response is called forth (the rapidity of the hemorrhage) and upon the ability of the heart to furnish the additional power demanded by the increased pressure. Thus in severe,

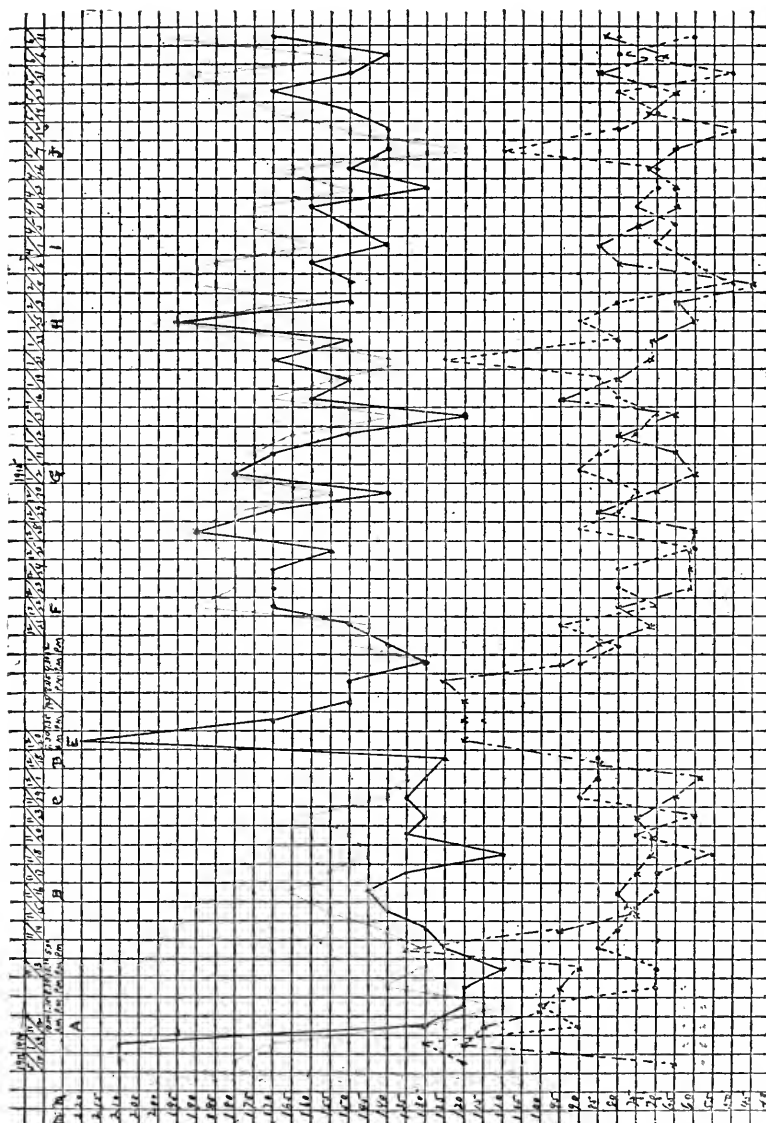


Fig. 40. (For explanatory note see page 221).

sudden hemorrhages there usually occurs coma, slow pulse, altered respiratory function and a markedly elevated systolic pressure, which bears a direct relation to the amount of and rate of development of the increased intracranial tension, whereas in cases where the compression is slow, as in brain tumor, the blood-pressure will not be materially affected, even though the amount of brain tissue eventually subjected to destructive compression be

FIG. 40.—Female. Aged sixty. Mitral incompetence of many years standing. Symptoms upon first examination chronic indigestion, palpitation, physical weakness, dizziness and dyspnea.

Observations recorded in 1912 and January, 1914, indicate the two highest observations of systolic pressure made during this period. At (A) shows observations made one-half hour after relief from attack of cardiac dilatation with pulmonary edema, unconsciousness, uncountable pulse, rapid respiration, etc. Relieved by hypodermic of morphine, atropin and nitro-glycerin. The small pulse pressure when first recorded was probably result of the overuse of atropin which did not, however, materially effect the improvement. Digitalis and strychnin were then substituted with the gratifying effect as shown between (A) and (B). During this time the patient was in bed.

At (B) there was another moderate attack of dilatation with pulmonary edema. Relieved by atropin and spartein hypodermatically. Between (C) and (D) the urine became very scant and albuminous, and at (E) another attack similar to the first occurred. This began while the patient was resting in bed and from no apparent cause. Similar measures occasioned a sharp drop as indicated, while cessation of hypodermatic treatment caused a rebound at (F) which was accompanied by an extremely high pulse pressure which did not however appear to affect the patient's improvement.

Between (F) and (G) sufficient cardiac recovery occurred to allow the patient to sit up for a short time. Cardiac irregularity developed here probably due to the use of digitalis, which was stopped and strychnin substituted.

(H) A mental shock caused the sharp rise in systolic pressure and pulse pressure, as shown, when the patient suffered a slight relapse.

(I) Cardiac asthma here became a prominent symptom and persisted with orthopnea until the termination, together with progressively increasing general edema.

(J) Patient over-exerted against advice and suffered symptoms very much like those of shock and in which the pulse pressure participated particularly. From this point pulmonary edema rapidly developed until the patient died suddenly.

far greater than in case of hemorrhage. Extremely high pressures may be encountered in acute hemorrhage, where readings of 400 mm. and over have been recorded.

Diagnosis.—The value of the blood-pressure test in the diagnosis of obscure cases of coma cannot be overestimated, as there is probably no other single test that can be quickly applied, which will give more valuable information. Repeated observations of the systolic pressure will indicate a cessation of the hemorrhage, as it is unlikely that a stationary or slightly falling systolic pressure will occur in the early hours of a cerebral hemorrhage, unless the destructive process has ceased. In regard to differential diagnosis, reports seem to show that hemorrhage into the anterior fossa affects blood-pressure least, while when into the posterior fossa (near the fourth ventricle) the greatest rise is noted. From a differential standpoint the blood-pressure test may also serve to distinguish a cerebral hemorrhage or an apoplectic coma from embolism, as in the latter the blood-pressure is not high.¹

Cheyne-Stokes Respiration.—Pollock² reports a series of blood-pressure estimations in fifteen cases of Cheyne-Stokes respiration arising from various causes, which confirm the earlier observations of Cushing that in Cheyne-Stokes respiration with increased endocranial tension, the blood-pressure is low during the period of apnea and high during that of hyperpnea, as well as the demonstration by Eysner that this fact is of clinical value in the differentiation of Cheyne-Stokes respiration with increased endocranial tension from other types. In the cases with in-

¹ H. D. Jump, *Int. Clinics*, vol. i, 21st Series, p. 49.

² *Arch. Int. Med.*, vol. ix, No. 4, 1912.

creased tension, the blood-pressure began to rise slightly before respiration commenced and began to fall after the summit of respiratory activity was reached, whereas in the other cases, the pressure began to fall after the beginning of respiration and rose as respiration diminished.

Diabetes Mellitus.—This condition, which is usually ascribed to perverted pancreatic function, manifests itself clinically through a series of symptoms caused by a state of toxemia, accompanied in a fair percentage of cases by cardiac and circulatory derangement. The latter manifests itself usually in cardiac hypertrophy and myocardial degeneration combined. Some cases show arterial and kidney sclerosis. This latter complication probably led early investigators to attribute high pressures to this disease. In the light of the combined statistics of more recent investigations it appears that low pressure is the rule, although a continued high pressure accompanies a minority of cases.

A. R. Elliott¹ studied a series of cases of diabetes mellitus with the following summary of results:

Number of cases observed.....	25
Males.....	13
Females.....	12
Average age of persons.....	45
Average weight of patients (pounds).....	156
Average systolic blood-pressure (mm. Hg.).....	127

In this summary the average systolic pressure is slightly misleading, since, in the thirteen patients who were under fifty years of age, the average pressure was 107 mm. Hg., while in the twelve cases over fifty years, the average was 150. If there is any relation between the amount of sugar and the level of blood-pressure, it would appear that a

¹ *Jour. A. M. A.*, xlix, No. 1, July 6, 1907.

high percentage of glycosuria favors a depression in systolic level. This is probably explained on the grounds that the resultant emaciation and brown atrophy or fatty change in the heart contributes largely to this condition. Acidosis also tends to a subnormal pressure.¹

As already intimated, the high pressure present in some cases, during the course of diabetes, can easily be explained by the presence of such complications as chronic nephritis, cardiac hypertrophy and arteriosclerosis.

Ehrmann,² considers blood-pressure records of particular value in determining impending coma, as in many instances he has noted that there is a marked decrease in the systolic pressure immediately preceding this phenomenon.

Mineral Poisons.—Lead Poisoning.—The effect of chronic lead intoxication frequently results in permanent changes in the arteries and kidneys, resulting in a secondary hypertension. There is, however, a form of hypertension occurring in lead poisoning, as evidenced by the typical colic, which is always accompanied by a moderate elevation in blood-pressure, which may remain elevated for several days, succeeding the attack (a primary hypertension).

With the knowledge of exposure to lead, followed by an attack of typical pain with high blood-pressure, we may be aided in difficult cases by the blood-pressure test to separate lead colic from renal and hepatic colic, in which the blood-pressure is low.

Acute Phosphorus Poisoning and Arsenic Poisoning—These are usually accompanied by a depression in systolic pressure.

¹ Ehrmann, *Berl. klin. Wochen.*, Aug. 4, 1913, No. 31, p. 13.

² *Loc. cit.*

Morphinism.—In the average case of chronic narcotic poisoning the blood-pressure, in the absence of cardio-renal complications, has been found to be below normal, although it is stated that occasionally a case is met in which the pressure ranges between 180 and 200, which is apparently caused by the attending constipation, as it is usually relieved by a brisk purge.

Momburg Constriction.—Dr. Fred L. Adair¹ has studied twenty-three cases in an effort to determine the effect of abdominal constriction by the Momburg tube on blood-pressure, pulse, etc. Cases showing abnormalities of the

ADAIR'S TABLE OF PULSE AND BLOOD-PRESSURE (MOMBURG CONSTRICTION)

Case	Pulse					Blood-pressure				
	Before	During		After		Before	During		After	
		Maxi- mum	Mini- mum	Maxi- mum	Mini- mum		Maxi- mum	Mini- mum	Maxi- mum	Mini- mum
1	95	97	97	88	88	116-118	132	122	118	112
2	104	103	103	106	106	144	148	90	144	124
3	86	96	96	94	94	114-116	114	114	116	116
4	84	120	110	72	72	122	126	98	128	118
5	86	104	98	92	84	146	154	138	146	130
6	86	78	72	92	74	128	166	138	128	110
7	88-102	122	116	98	98	144	148	111	140	140
8	98-120	136	120	96	88	124	110	100	124	110
9	96	104	80	120	80	120	140	120	120	96
10	88	92	88	72	72	126	144	126	122	120
11	84	120	92	88	84	124	136	130	126	92
12	86-104	160	116	135	90	125	175	154	128	100
13	66	120	72	112	56	106	136	128	106	70
14	64	84	58	96	60	110	128	122	124	90
15	70	116	64	104	56	116-118	184	158	122	110
16	84-88	120	100	128	80	118	156	102	136	114
17	72	132	110	88	64	120	166	160	150	120
18	64	112	96	88	64	110	120	114	124	116
19	80	96	68	?	?	110	116	108	?	?
20	76	104	76	100	64	107	115	104	?	?
21	98	132	114	88	72	136	182	157	128	116
22	70	92	84	84	60	110-112	126	115	114	110
23	82	92	84	84	72	138-140	166	156	132	124

¹ *Surg., Gyn. and Obstet.*, 1912, p. 112.

heart, blood-vessels or kidneys were excluded and all observations were made in the supine posture without anesthesia. While of necessity the duration of application was short, the femoral pulse was always obliterated. The detail findings of this series are shown in the accompanying table, and correspond in general with the results of earlier observers, notably Wolff. The most dangerous period appears to be when the tube is removed, and this is most dangerous in those presenting arterial change, cardiac disease, anemia and vasomotor instability.

Polycythemia.—Moller¹ states that in his experience and from a study of literature, there is no direct relation between the level of systolic blood-pressure and the number of red blood corpuscles. In his study of thirty-five patients, all with systolic pressure of 180 mm. or above, two only showed an abnormal proportion of red corpuscles. On the other hand, we know that in conditions accompanied by anemia, in the absence of arterial and renal change, it is usual to find subnormal pressures.

Pleural Effusions.—Large effusions tend to increase systolic pressure while their removal is accompanied by a fall. Probably on account of the proximity to the great vessels and the greater rigidity of the thorax as compared with the abdominal cavity, pleural effusions occasion a greater rise than do ascitic. This rise in pressure should be taken into consideration when weighing the possibility of the presence of a coincident nephritis. During the removal of fluid, one may expect a greater fall in recent than in old effusions. The usual fall is from 15 to 25 mm. When observing the blood-pressure during the re-

¹ S. Moller, *Deutsch. med. Wochen*, xxxiv, Oct. 29, 1908.

moval of fluid, a sudden fall accompanied by weakness, palpitation, vertigo or signs of collapse should indicate the need for a very slow withdrawal of fluid if not an immediate cessation.

This dangerous fall in pressure may be combated by the application of a suitable abdominal binder which, by increasing intra-abdominal pressure, counteracts the sudden change in pressure surrounding the great vessels; trouble may also be averted by changing the posture of the patient to a reclining one.

Prolonged Epistaxis Associated with Increased Vascular Tension.—Girard H. Cocks¹ and Harold Hays² have noted the frequent association of prolonged and profuse epistaxis with high blood-pressure. Hays states that these sudden and profuse hemorrhages are usually associated with one of two classes of circulatory disease.

1. Arteriosclerosis involving the arterial system and the myocardium.

2. Valvular disease, or congenital deformity of the heart.

In the first group, the epistaxis seems to be the direct result of high arterial tension, and is both a warning sign of impending apoplexy, and a beneficial act on the part of nature to relieve a dangerously high blood-pressure. This fact should lead to inquiry into the state of the circulation, particularly in all persons of advancing years, who show a tendency to epistaxis, especially if uncontrollable by the usual means. Relief from both the loss of blood and the danger attending a markedly elevated pressure may best be accomplished by measures directed to-

¹ "The Laryngoscope," January, 1911.

² *N. Y. Med. Jour.*, Mar. 4, 1911.

ward controlling the hypertension. This in Hay's experience is best accomplished in emergency by large doses of morphia.

Renal and Biliary Colic.—Abdominal pain accompanying these two conditions has no effect upon blood-pressure, unless obscured by a complicating nephritis. This fact should help to differentiate them from tabes and from lead colic, both of which give a marked hypertension.

Shock.—(See Surgery, Chapter XX).

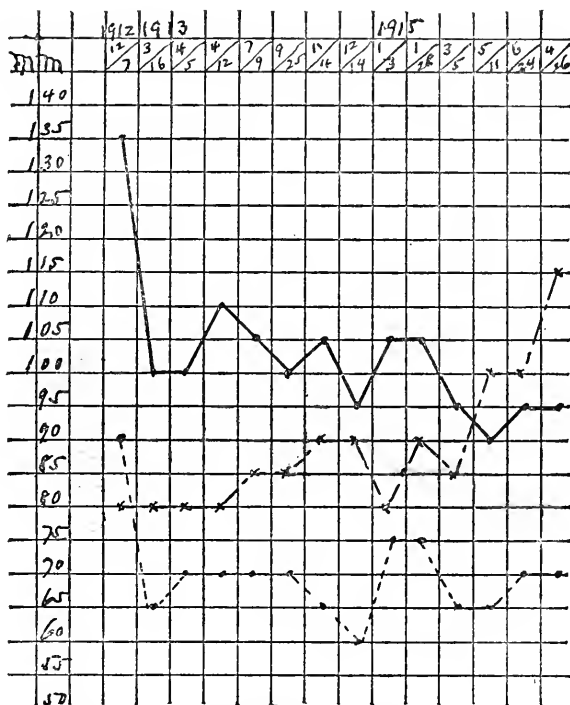
Thoracic Aneurysm.—In thoracic aneurysm the pulsus differens may be definitely determined by the blood-pressure test, taken upon both arms, whereas when the finger is employed one may be greatly misled by the findings. As an example of this, in one case of undoubted aneurysm of the last third of the arch of the aorta the left radial seemed distinctly smaller than the right, and the signs and radiograph showed an aneurysm located apparently so as to interfere with the flow of blood through the left subclavian, but the sphygmomanometer showed an average of 5 mm. higher on the left side and an autopsy showed the sac just below the subclavian.

In the differential diagnosis between thoracic aneurysm and dilatation of the arch of the aorta, O. K. Williamson¹ says the latter shows a greater increase in blood-pressure than the former, and if the difference in pressure in two arms is 30 mm. or more, it speaks strongly for aneurysm. Between aneurysm and mediastinal tumor a difference between the two sides of 20 mm. or more indicates aneurysm. While these reports as far as I know, have not been confirmed, and as I have had no experience in the matter,

¹ *Lancet*, Nov. 30, 1907.

they must be taken with some question, but may prove of value in aiding the elucidation of difficult cases.

Hodgkin's Disease.—The etiology of this disease is still obscure, the chief theories being that of a specific infection



toms a marked reduction in systolic pressure and in pulse pressure, and because of the comparative rarity of the disease the chart (Fig. 41) has been introduced.

NEUROLOGIC DATA

As would naturally be supposed both functional and organic affections of the central nervous system produce definite and easily demonstrable effects upon the circulation and blood-pressure.

Neurasthenia (See Hypotension, Chapter VIII).—Neurasthenia or the fatigue neurosis resulting from lack of nervous energy and instability of the sympathetic nervous system is naturally, when uncomplicated, accompanied by hypotension. We may include under this head the psychic instability of blood-pressure, so beautifully discussed by Schrump¹ where he shows that before we may arrive at a decision that a low blood-pressure is pathologic, we must make sure that it is not psychogenic. He also makes the interesting statement that a rise in pressure of psychogenic origin affects chiefly the systolic pressure; as the mind does not seem to have an influence upon the diastolic pressure, which is unaltered. Psychic instability is almost constantly present, in all individuals to some degree, but is much more marked in the neuropath. It is sometimes difficult to determine by one examination a normal from a pathologic alteration in blood-pressure, and it may become necessary to divert the patient's attention and to repeat the test at a subsequent time. Furthermore, it must not be overlooked that the period of absolute rest which

¹ *Deutsch. med. Wochen.*, Dec. 22, 1910.

usually begins the treatment of grave neurasthenia, is itself a cause for a lower blood-pressure. The degree to which the pressure falls in this condition depends somewhat upon the

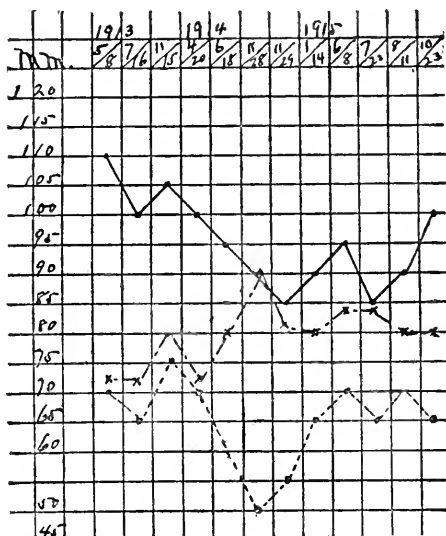


FIG. 42.—Female. Single. Aged forty-one. Typical neurasthenia with ever-changing symptoms. Lacks initiative and is morose. Constantly worrying about her condition. Phthisophobe. Many kinds of treatment have been only temporarily successful. Elevation of pressure as shown in the first few observations probably result of mental excitement. This chart shows well the depressed systolic pressure, which in spite of measures directed toward restoring normal pressure fails to respond, and the extremely variable pulse pressure, which under usual conditions is smaller than normal.

The reduction of pulse pressure tends to follow the general physical condition of the patient, yet in spite of the small pulse pressure this patient at no time shows either renal or circulatory embarrassment.

gravity of the disease and the temperament of the patient, but is usually moderate.

I have been unable to find any reference to a hypotension lower than 80 mm. systolic in neurasthenia. See Fig. 42.

The treatment of this disease when successful may be

indicated by a gradual return of the pressure to normal. It must be borne in mind that complicating nephritis may so affect the blood-pressure as to render the findings of no value.

Epilepsy.—According to Fisher,¹ in patients over forty there is a general tendency toward an elevation in pressure which is not usually seen in younger cases. During the paroxysm authorities agree that the systolic pressure may rise to a great height and remain high throughout the attack, falling to an abnormally low level during the subsequent stage of depression. This point may be of value in separating an epileptic from an uremic attack, as the post-epileptic fall does not occur in cases of uremic origin.

Tabes Dorsalis.—Lewellys F. Barker recently reported some cases of this disease in which the blood-pressure varied between 190 and 215 mm. Hg. Other authors have had similar experience, noting the rise usually during the paroxysm of abdominal pain. Jump² calls attention to this important differential point, that while with abdominal pain in gastric crises of tabes the blood-pressure is nearly always markedly elevated, it is usually low or normal in renal or biliary colic.

The common occurrence of arteriosclerosis in tabetics does not appear to be a cause for continued high pressure, which in the majority of cases is normal between the crises. It has been noted by some observers that the pressure rises some time before the pains begin, which fact, if detected by means of the sphygmomanometer, may be used therapeutically to ward off the impending attack.

¹ Edward D. Fisher, *Jour. A. M. E.*, Aug. 3, 1912, lix, No. 5, p. 395.

² *Internat. Clinics*, Vol. i, Series 21, p. 49.

Mania.—Hawley¹ reports certain alterations in blood-pressure in maniacs, which he considers as typical of the different stages of this affection. Thus both the systolic pressure and the pulse pressure increase as the restlessness of the patient becomes more marked, to decrease again as the patient recovers from the attack. Arteriosclerosis existing in maniacs accentuates this rise, which is generally then maintained during the intervals of quiet and depression.

In stuporous cases the systolic pressure and pulse pressure are usually below normal and are accompanied by a slow pulse. In depressed cases the systolic pressure is low and the pulse pressure small, but not so low as is usually encountered in stuporous cases.

Melancholia.—In melancholia the average systolic pressure and the pulse pressure remain at or near normal as long as there is no muscular resistance, or no other factor such as arteriosclerosis to produce a rise.

Paresis.—In the majority of paretics the systolic pressure tends to a lower level than in normal individuals, although this is by no means the rule, because of the frequency of arteriosclerosis with its cardiac and its renal accompaniments. Walton² compared two groups of cases, the first showing the complications just referred to, while in the second group there was no record of arterial, cardiac or renal disease.

Group I		Group II	
Age	Systolic	Age	Systolic
Under 40.....	125 mm. Hg.	Under 40.....	115 mm. Hg.
40 to 50.....	129 mm. Hg.	40 to 50.....	mm. Hg.
Over 50.....	147 mm. Hg.	Over 50.....	118 mm. Hg.

¹ M. C. Hawley, *Arch. Int. Med.*, November, 1913.

² G. L. Walton, *Jour. A. M. A.*, Oct. 27, 1906, xlvii, 17.

Schmigergeld¹ briefly summarizes our knowledge of blood-pressure in general paresis as follows:

1. The blood-pressure in this disease is variable.
2. In the absence of complications the pressure in the majority of cases is subnormal.
3. There appears to exist no relation between the mood of the paretic and the state of the blood-pressure.

¹ *N. Y. Med. Jour.*, Aug. 28, 1909.

CHAPTER XV

HYPOTENSION

Definition.—This term is employed to designate alterations in arterial blood-pressure in which the systolic blood-pressure curve maintains an average level below the established normal minimum. The actual level of this pressure will be affected to some degree by the age and other physiologic factors, which control the normal level of pressure (see page 42).

The Lower Normal Limits.—The limits are, of course, largely arbitrary, depending as they do upon so many variable and varying factors. To maintain their full value, they must be modified to conform to our knowledge of the many so-called physiologic factors active in each individual case (see Chapter VI).

Experience teaches that 105 mm. may be taken as the low limit of normal blood-pressure in young men, and 95 mm. as the normal low limit in young women. This will of necessity be modified slightly by the age, occupation and muscular development of each individual. The only way to estimate the degree of abnormality in the blood-pressure is to apply the knowledge obtained from experience in examining a large number of cases. Therefore it is usually advisable to employ the blood-pressure test as a routine in all cases, in order to develop one's ability to interpret the significance in each individual case.

Unfortunately, literature almost entirely lacks exhaustive

studies of low systolic pressures, with the exception of certain definite conditions, as acute and chronic infections, shock, hemorrhage, Addison's disease, etc., where the references are many and the data conclusive.

Varieties of Hypotension.—We must admit the possibility of a relative hypotension, in which the curve of pressure, while above the established normal is yet so far below a previous long-continued high pressure, that it presents the physical phenomena of a pathologic low pressure. This point is discussed more fully below.

Hypotension is also encountered as a coincident phenomenon of many pathologic conditions in which it is usually the result of the effect of some substance or substances affecting the cardiac output, the arterial coats or the vasomotor mechanism. These include infections, certain metabolic diseases, shock, hemorrhage, etc. Such conditions occasion a temporary hypotension, which is relieved, as the patient recovers or is relieved of the metabolic fault. These conditions will be considered in detail in appropriate sections and need not now be discussed.

In order to fully comprehend the discussion which follows, some form of clinical classification of low blood-pressure must be formulated. The following seem to be the most satisfactory subdivisions:

(a) Terminal hypotension, (b) essential hypotension, (c) relative hypotension and (d) temporary or secondary hypotension.

(a) **Terminal Hypotension.**—The term is used to indicate the more or less marked fall in systolic pressure, usually accompanied by a diminishing pulse pressure which indicates the approaching end of life (see Fig. 43).

Usually before death, irrespective of the cause, the blood-pressure tends more or less rapidly toward zero. The rate at which this occurs and its relation to the actual cause of death, is determined by so many factors about which almost nothing is known, that as yet little may be said with

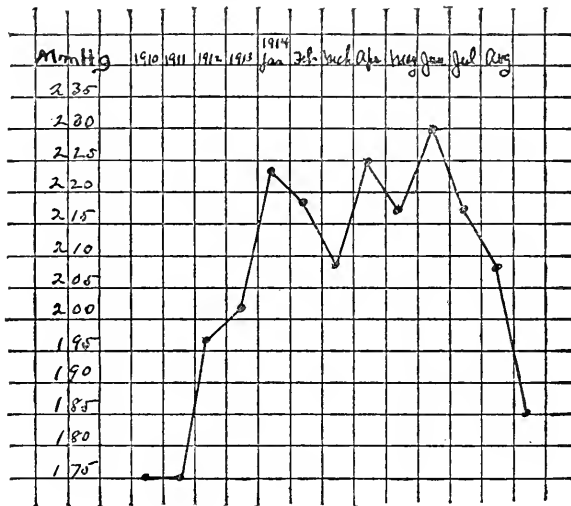


FIG. 43.—Chronic interstitial nephritis. Male. Aged fifty-six. This chart shows the rapidity of the rise in systolic pressure in a case of chronic interstitial nephritis and myocarditis in a patient of fifty-six who failed to appreciate the necessity of care or treatment and was willing to, as he said “take his chances.” Cardiac dilatation began when the systolic pressure reached 230 and rapidly advanced. The patient died in an attack of pulmonary edema with systolic pressure 185. Observations before 1914 recorded on chart are yearly averages. 1914 observations show average systolic pressures by months.

certainty. According to Janeway, in protracted illness pressures as low as 60 mm. (5-cm. cuff) may exist for several days before death (note). In such cases the hypotension may be of some value as a sign of impending dis-

NOTE.—I have seen a case of acute bichlorid poisoning in which a systolic pressure of 65 was recorded several days before death.

solution, but as a rule the terminal fall in pressure is usually a matter of hours or even minutes. Occasionally the fall is too rapid to be observed and an apparently normal systolic pressure may be demonstrated almost up to the moment of death.

(b) **Essential Constitutional or True Hypotension.**—*Definition.*—This type of hypotension may be defined as that form of lowered systolic blood-pressure met in a certain class of individuals who, though not distinctly ill, do not show evidences of robust health. These persons do not seem to have the physical development, or circulatory power to maintain normal blood-pressure values even under the most favorable circumstances. Systolic pressures of 100 or lower are the rule (see Fig. 42, page 231).

Since the pioneer work of L. F. Bishop in this field, a few spasmodic efforts have been made to assign a definite symptomatology to this condition, and recently Goodman¹ has contributed a valuable paper upon this condition.

Symptomatology.—These cases are not actually ill, nor are they ever well, but complain of all sorts of symptoms which can be traced directly to low pressure as the only assignable cause.

Such individuals are unable to withstand any prolonged or severe physical exertion without fatigue.

They have not only a habitually low pressure but they also respond very poorly and uncertainly to measures directed toward improving the circulation or to those stimuli which in the normal person result in a rise in pressure.

The pulse pressure is usually smaller than normal, indi-

¹ E. H. Goodman, *Am. Jour. Med. Sci.*, April, 1914.

eating a defective cardiac output, while there is no relation between blood-pressure and pulse rate.

These cases are usually associated with enteroptosis and may have a tendency to attacks of temporary dizziness, upon sudden change of posture; many are undoubtedly of intestinal toxemic origin. Constipation tends to keep the pressure down, as does also a marked indicanuria, which is so frequently encountered in these cases. Headaches are frequent, and are often relieved by measures directed toward increasing the pressure. These cases wake up tired, then improve during the early hours of activity, only to become exhausted long before the end of the day.

Many are thin and poorly nourished, and suffer continually from chilly hands and feet, which are often moist and may be actually wet. The dependent hands are bluish and when one is elevated to above the head for a short time it becomes abnormally pale.

Those patients lack initiative, and every movement seems to be an effort; even when shaking hands, it is done in a half-hearted manner, and life is generally viewed through dark glasses. They are decidedly of the depressive neurasthenic type. Mental tire is often extreme in spite of the evidently active mind. Some of these cases undoubtedly belong to the class of congenitally small hearts and arteries.

(c) **Relative Hypotension.**—This term would seem to be a necessary one and should be applied to those cases whose actual pressure, while still above the estimated normal, has fallen from a former pathologic high level to such a degree that symptoms due to the fall have developed. A fairly common example of this is the frequent occurrence of edema

or other signs of circulatory failure following *injudicious attempts to reduce a high pressure*.

The same condition obtains in a failing cardiovascular system, when the pressure has been for a long time high. (See chart, Fig. 44.) Here also we may have most serious and distressing symptoms, pointing to circulatory failure, and yet the pressure may be found still above the estimated normal level.

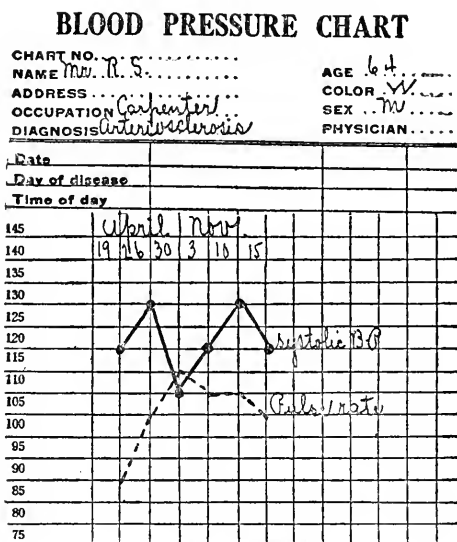


FIG. 44.—Hypotension of lost compensation (relative hypotension). Arteries markedly sclerosed, heart showed myocardial degeneration, pulse always rapid. Cerebral symptoms marked, treatment had very little effect.

(d) **Secondary or Temporary Hypotensions.**—All other cases presenting an abnormally lowered systolic pressure should at present be placed under this head. Etiologically these cases will be found singly or combined to be due to cardiac, vasomotor, actual muscular or of blood volume or composition changes.

G. M. Piersol¹ has conveniently grouped the several causes as follows:

1. Hypotension due to vasodilatation, shock and collapse from whatever cause.

2. Acute infections and toxemias.

3. Hypotension due to cardiac weakness, as in chronic myocardial degeneration, acute dilatation, arteriosclerosis and chronic nephritis.

4. Orthostatic albuminuria and amyloid disease, not associated with true nephritis.

5. Advanced cachexias, occurring in the course of carcinoma, diabetes and nephritis.

6. Hypoplasia of the chromaffin system as in Addison's disease.

7. Hypotension due to diminished blood volume as in cholera, persistent diarrhea, dysentery, cholera infantum, Graves' disease and hemorrhage.

8. Certain nervous affections, of which paresis and epileptic coma are examples, also true neurasthenia.

9. Drug poisons, as tobacco. This ordinarily raises blood-pressure, with the apparent anomaly that heavy smokers frequently have subnormal pressures. For the effect of drugs on blood-pressure, see Chapter XXIII, page 409.

10. There are many examples met with of the so-called gouty or rheumatic manifestations of lumbago, sciatica, or neuritis which show a blood-pressure somewhat below normal. Many of these cases have a subnormal acidity of the urine, and are liable almost constantly to a copious deposit of phosphates which leads to, or is accompanied by, a state of nervous depression.

¹ *Penn. Med. Jour.*, May, 1914, xvii, No. 8.

Extreme Low Pressure.—The lowest blood-pressure in an adult compatible with life has been reported by Neu to be from 40 to 45 mm., and this only occurred with sub-normal temperature accompanied by unconsciousness. He has observed and recorded recovery after a temporary fall in pressure as low as 50 mm.

Pressures of 80 to 95 are not uncommon, and may persist for long periods, to be followed finally by a recovery of normal values.

Effects and Danger of Hypotension.—The direct effect of a falling blood-pressure is the accumulation of an abnormal amount of blood in the veins, and a slowing of the current in the arteries. This will affect the capillary circulation and interfere with the nutritive and secretory processes which depend upon it. The most serious effect is on the heart, as it has been shown that complete loss of vasomotor tone soon leads to death, because of the gradual accumulation of nearly all the blood in the body on the venous side, so that the heart has no blood upon which to act.

CHAPTER XVI

HYPERTENSION; HYPERPIESIS

The term hypertension, is generally applied to any condition in which the blood-pressure is maintained at a level above normal, irrespective of the degree or duration of the elevation. Such use of the term admits of confusion and doubt in the mind of the reader, because of the diversity of conditions included in so comprehensive a term. The fact that careful clinicians continue to report cases with hypernormal blood-pressure, in the absence of either cardiac or nephritic degenerative changes, and since we are able to obtain a permanent and lasting return to normal blood-pressure values in certain cases by appropriate treatment and hygiene, it would seem to justify an effort to limit this term and to admit the presence of such a clinical entity, having as a prominent symptom, a transitory elevation of systolic pressure; for if we fail to recognize this clinical condition, there will remain an unfilled gap in our clinical conception of cardiovascular and renal pathology.

That we may accept this condition as a pathologic and clinical entity, is shown by the fact that its existence is recognized by many modern authorities, among them Janeway,¹ Butler,² R. A. Torrey,³ T. C. Janeway,⁴ and H. W. Cook.⁵

¹ *Am. Jour. Med. Sci.*, 1906, cxxxi.

² *The Practitioner*, 1909, lxxii.

³ *Penna. Med. Jour.*, April, 1914.

⁴ *Jour. A. M. A.*, Dec. 14, 1912, lix, p. 2106.

⁵ *Jour. A. M. A.*, Jan. 28, 1915, lxiv, No. 4.

Definition of Hyperpiesis or True Hypertension.—It would seem best, at least for clinical purposes, to limit the

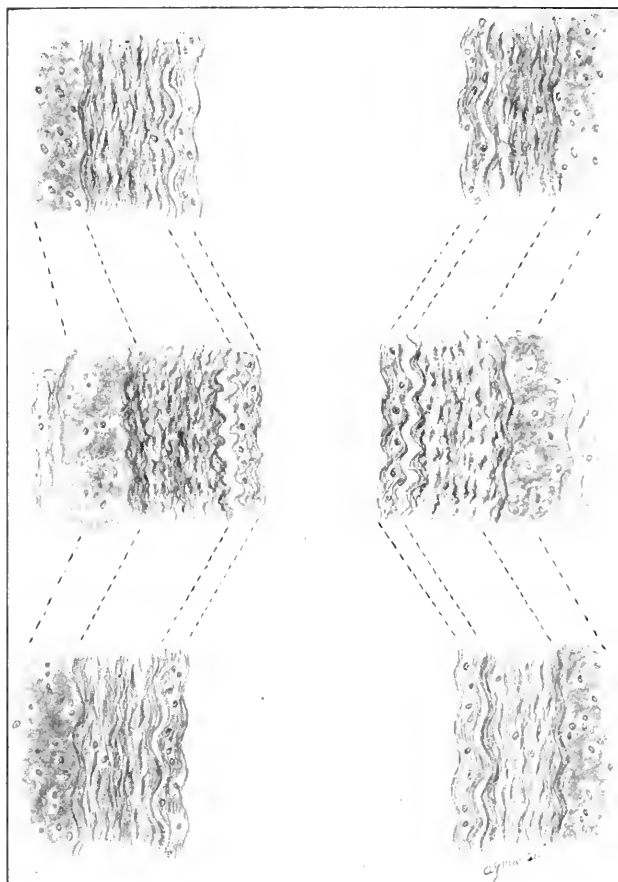


FIG. 45.—Hypertonia vasorum; upper line, section of normal artery middle, section of contracted artery; lower, section of artery restored to its natural partial contraction. (L. F. Bishop, in *Medical Record*.)

term hyperpiesis or true hypertension, to a well-recognized symptom-complex in which the rise in systolic pressure is moderate and persistent, but not permanent, and to

designate all other high pressures, which are either dependent upon, or accompanied by distinct and clinically recognizable arterial, kidney or heart changes, as *high blood-pressure*. Thus we limit the term hypertension to an elevation of blood-pressure dependent largely, if not solely, upon a muscular change in the arterial walls and

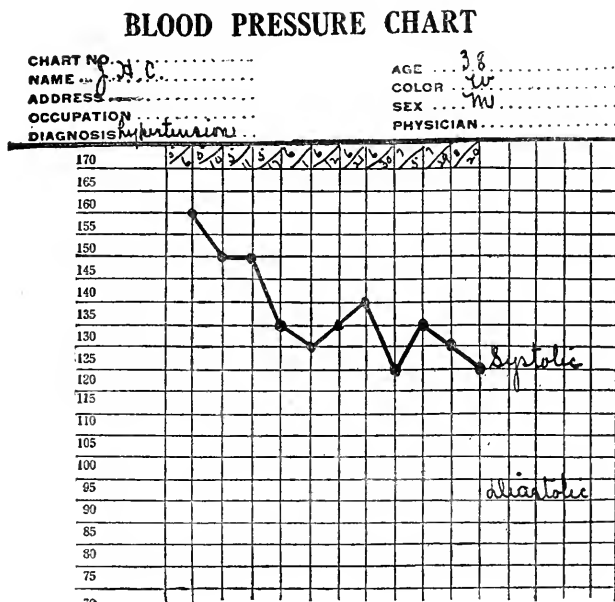


FIG. 46.—Illustrates pure hypertension and the effect that may be expected to follow measures directed toward relief of underlying toxemia.

capillaries¹ whereby they are temporarily narrowed and contracted. This corresponds to the "Hypertonia Vasorum" of Bishop² given in his classification of arterial disorders, and (see Fig. 45) which may be overcome by ap-

¹ Wm. Russell, "Arterial Hypertension, Sclerosis and Blood-pressure," Philadelphia, 1908.

² L. F. Bishop, *Med. Rec.*, Dec. 4, 1909.

propriate treatment (see Fig. 46); as contrasted with true high pressure, where, in addition to other conditions, there is a permanent pathologic change in the arterial coats, either in part or as a whole, and which is never entirely overcome by treatment.

By adhering to this distinction we are immediately enabled to appreciate the inherent pathology of this condition, and also to understand the different results obtained by methods directed either toward its relief, or the relief of the resultant symptoms.

Etiology.—Regarding the cause or causes of hypertension in its restricted sense, these may be summed up in the word “intoxication,” although much yet remains to be explained concerning the precise nature of the processes leading to its production. The causes are essentially the same as those producing arteriosclerosis, but differ in that they have acted over a shorter period of time and are probably less active in character. This belief is borne out by the clinical fact that, if permitted to continue, the condition gradually progresses until it imperceptibly merges into and becomes one of true arteriosclerosis and cardio-renal disease.

That the adrenals and other members of the chromaffin system play an important rôle is shown in various ways. Recently Sergent and Cottintot¹ have shown that irradiation of the adrenals in eleven cases of hypertension, in which all known causes of this condition had been eliminated, caused a reduction in systolic pressure of from 40 to 50 mm. Hg. Obesity, which is considered by some observers to be another phase of disturbed internal secretion,

¹ *Jour. A. M. A.*, Mar. 28, 1914, lxii, 13, p. 1032.

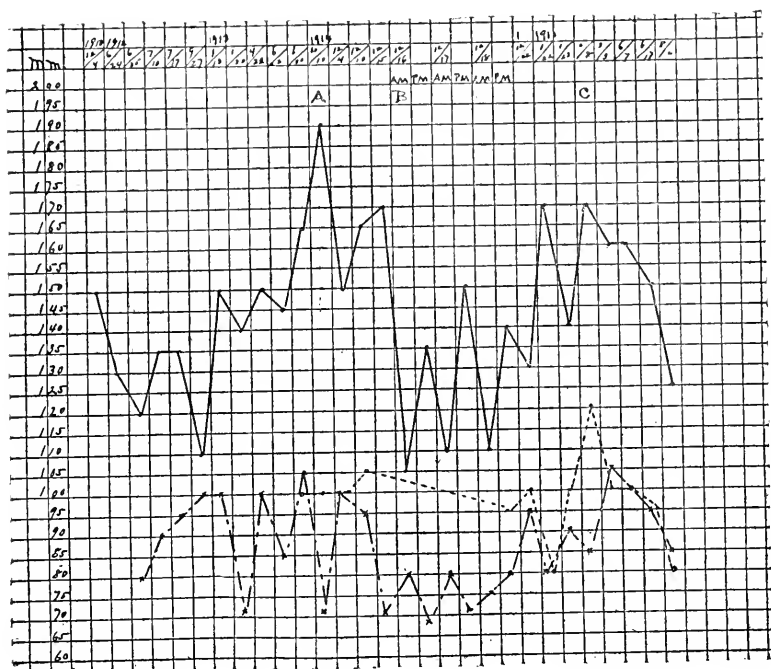


FIG. 47.—Hypertonus. Female. Married. Aged thirty-three. Has always enjoyed good health. Very energetic. Has a tendency to overweight and laces very tightly. The rise in pressure (A) is wholly unexplained. There being no corroborative findings, which would suggest nephritis. This is probably a case of pure hypertonus which, if unarrested, will gradually merge into one of permanent cardio-renal disease. The remission in pressure following (A) was accomplished by hygienic means, while the morning-evening observations (B) were made before the patient arose in the morning and in the evening after an ordinary day's occupation. Tight lacing and nervous temperament are undoubtedly factors in this case. (C) Phthalein test at this date resulted as follows: 1st hour 40 per cent., 2d hour 20 per cent., 3d hour 10 per cent. Total 70 per cent. Urinalysis usually showed albumen varying from a trace to a moderate amount, a normal specific gravity, and occasional cylindroid and hyaline casts.

has been suggested by C. Fessinger¹ as another possible cause of this condition, because in thirty out of 160 persons of excessive weight showing hypertension, he was able to eliminate all other probable causes excepting overweight.

Schlayer² advances the plausible theory, and presents an array of evidence to sustain it that hypertension when persistent is due to a condition in which the arterial system has become sensitized by some means, rendering it hypersusceptible to epinephrin or other similar substances and to other toxins which may be circulating in the blood. The source of this sensitization may be in the kidney, even though the kidney changes when present are usually a parallel phenomenon. Clinical observations seem to sustain this assumption, explaining at the same time why simple rest and increased elimination have such beneficial influence on the condition.

Provocative Causes.—These are the same as those which enter largely into the production of arteriosclerosis and its complications, differing only in that they are more mild in their impression, and act intermittently or have a shorter duration. These are considered more fully under the head of Arteriosclerosis, to which subject they properly belong (see page 261). Suffice it to say here that it is generally recognized, from both clinical and pathologic standpoints that the etiologic factors are the same, and that the conditions met, hypertension only or high blood-pressure, will depend largely upon the susceptibility of the individual (see Fig. 47), the activity of the toxic agents and the duration of their influence.

¹ *Bull. de l'Acad. de Med.*, Apr. 16, 1912, lxxvi, 16.

² *Münch. Med. Wochen.*, Jan. 14, 1913, lx, No. 2.

Symptomatology.—Hypertension is a subtle condition often lurking where least suspected. There may be no change in the palpable arteries except a barely distinguishable narrowing and stiffening when rolled under the finger, a hardly noticeable change in the aortic second sound, possibly a faint systolic whiff at the aortic cartilage, while the urine remains practically normal. The systolic blood-pressure will be found to be from 140 to 180 mm., depending upon the degree of arterial contraction.

By many, a progressive change in the arteries and a gradually rising blood-pressure are looked upon as normal conditions in late middle life or in early old age.

Hypertension is, I believe, always a sign of the beginning of a pathologic change, which according to Huchard, Russell and others is the danger signal, a warning that some alteration must be made in the daily life, or the primarily curable condition will progress and merge into cardiovascular-renal disease (see Fig. 48).

A permanent increase in blood-pressure in the young adult, or in early middle life, in the absence of discoverable organic change in the heart, blood-vessels or kidneys, is always a sign of chronic toxemia; a poisoning arising from some error in metabolism or deficiency of elimination either intestinal or urinary or both. Such a rise in blood-pressure is rarely discoverable, except by the routine estimation of blood-pressure of those coming under the physician's observation either as patients or for examination for life insurance. In the early stages symptoms referable to the condition either must be rare, or if mentioned are attributed to overwork, mental worry, neurasthenia, etc. On the other hand, by careful questioning suggestive symptoms

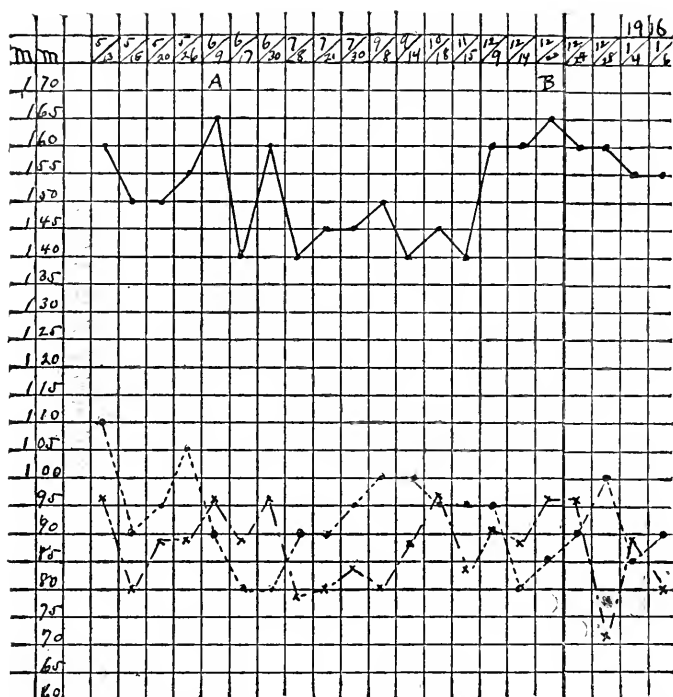


FIG. 48.—Toxic Migraine. Male. Aged forty-three. From a member of a family in which typical migraine headaches are common, having occurred in the patient's father, mother, aunt and sister. In this patient these attacks come on with great regularity, at seven- or eight-day intervals. When first seen these headaches were causing great annoyance by interfering with his daily activities. Refractive errors have been corrected. The Wassermann reaction is negative. The patient is of sedentary habits and is a large eater. His work is purely mental. The patient is constipated and the urine, upon frequent examinations showed evidence of intestinal toxemia. Therapeutic efforts were directed solely toward elimination of toxemia and resulted in considerable relief until the patient at (A) returned from a ten days' business trip. During this time he had several headaches and a number of dizzy spells. Dietetic measures were again instituted with the following result, namely, a moderate decrease in systolic pressure with a great fall in pulse pressure incident to an approximation of systolic and diastolic pressures. The rise at (B) seemed to be incident to a return of constipation which was relieved by exercises.

may be elicited, such as fleeting dizziness, throbbing temples, tinnitus aurium, disturbed sleep, cold hands and feet, gastric distress and flatulency, constipation, lack of interest and of power of concentration, diminished desire to be up and doing, distaste for physical exertion and weakened tolerance for substances which affect the brain, such as alcohol and tobacco.

The blood-pressure does not need to be greatly increased in order to injure the heart and cause permanent change in the blood-vessels and in the kidneys. The additional amount of work required of the heart to overcome the resistance of a few millimeters of mercury mounts up surprisingly (see page 275).

Concerning the actual systolic pressure level encountered in hypertension as contrasted with that found in cases where arteriosclerosis and degenerative changes have developed, most authorities agree upon 160 to 170 mm. Hg. as the dividing line, although this is by no means arbitrary, since it is well known that well-developed cases of arteriosclerosis and nephritis may exist with systolic pressures averaging lower than 170, and that apoplexy has occurred in patients who have never shown a systolic pressure of over 170.

The diastolic pressure will be found elevated, but to a less degree than the systolic pressure, this giving a moderately increased pulse pressure, but the difference is rarely as great in proportion as shown by the pulse pressure usually met in arteriosclerosis and nephritis. A typical case might present the following: systolic pressure 160, diastolic pressure 100, pulse pressure 60. It would be an error to base a diagnosis upon this alone as only repeated study

and the careful weighing of all evidence, permits the arrival at a safe and correct opinion.

Diagnosis.—This requires fine judgment, is always difficult and becomes additionally so as the boundary line between hypertension and arteriosclerosis is approached.

It will be a great aid to diagnosis to keep the fact constantly before the mind that hypertension is a cardiovascular (largely vascular) function of a temporary nature, which we now recognize and correct, while arteriosclerosis is a permanent pathologic change of the tissues of the arterial walls for which we know no remedy.

The eliciting of a group of otherwise unexplainable symptoms as enumerated above, in the absence of demonstrable (after careful and repeated search) lesions of the heart and kidneys, and with only a moderately elevated systolic pressure (below 170) and a slightly increased pulse pressure, in persons susceptible to auto-intoxication, as those of sedentary habits or accustomed to overeating, especially when blood-pressure reducing measures rapidly reestablish normal blood-pressure values, should afford the clinician a reliable diagnosis. A note of warning should be sounded here, which is that one should not be too ready to make a positive diagnosis of pure hypertension, as it is safer to err on the side of the more grave condition and so proceed in the treatment. This will avoid a too sanguine view of a possibly serious case which at first being considered trivial, later develops into one of true arterial or renal disease.

Syphilis is well recognized as a cause of hypertension; it acts in the same manner as other circulating toxins and

therefore may be looked for as one factor in the production of hypertension (see also Chapter XIII).

Concerning the relationship between true hypertension and permanent renal and arterial changes, we are coming more and more to the view that high blood-pressure is primary and the involvement of the kidneys and arteries only incidental.¹

This seems only natural when we realize the great surface area of arterial walls which are exposed to the action of irritating substances in the circulating blood, as compared with which the kidneys actually only receive a small portion of these toxic irritants.

¹A. W. Hewlett, *Jour. A. M. A.*, May 25, 1912, lviii, No. 21.

V. Bie, *Ugeskriftn f. Sarger.*, Mar. 4, lxxxvii, No. 9.

Cottentot and Sergeant, *Jour. A. M. A.*, Mar. 28, 1914, lxii, No. 13, p. 1032.

CHAPTER XVII

ARTERIOSCLEROSIS

In any consideration of this subject we should primarily insist upon an absolute distinction between hypertension and arteriosclerosis. Arterial hypertension is a cardiovascular phenomenon having perversions and abnormalities which the diagnostician can now recognize and in many instances successfully arrest and correct (see Chapter XVI, page 243). Arteriosclerosis is a permanent pathologic change of tissue for which there is no remedy. The source of danger in arteriosclerosis is not primarily in the thickened artery but in the amount of permanent blood-pressure elevation accompanying it. The thickened artery itself is a physiologic development of a protective nature and represents a factor of safety rather than a source of danger.

Latterly our conceptions concerning the origin and development of arteriosclerosis have undergone great changes, largely due to the fact that during the past few years much experimental work has been carried on and many clinical investigations made, which in great measure prove the causative relation of a large number of exciting agents, the effect of which, either directly or indirectly, on the vessel walls is the provocative cause of arteriosclerosis. In another direction, studies have demonstrated the relation of this condition to abnormally and persistently high

blood-pressure and to cardiac and renal diseases (see Chapter XIX and Chapter XVIII).

While admitting that in the present incompleteness of our knowledge on this subject, final and conclusive proof of the relation of the various agents, supposed to be fundamentally responsible for the development of vascular and renal sclerosis is still lacking, because they are extremely difficult and in many cases impossible of demonstration, nevertheless it would seem highly desirable, from the clinical standpoint, to accept the data at hand (both clinical and pathologic), and for the present at least to separate and to consider individually, hypertension, arteriosclerosis, cardiomuscular and renal diseases, as causes of persistent high blood-pressure and, by a careful analysis, attempt to establish their predominant etiologic relation to changes in the arterial wall, the heart muscle and the kidneys, as the case may be, in each case of high pressure studied.

In the following pages the purely scientific investigator should not be too critical of the conclusions drawn, but should allow the author some latitude in dealing with this still open subject, as, throughout, the chief effort has been to reduce a very large and complicated subject to a practical working basis, one that may be applied to conditions met in daily practice. After all, this is the chief concern of the practitioner.

PHARMACODYNAMIC STUDIES OF THE CAUSE OF ARTERIOSCLEROSIS

Much of the experimental work in the study of arteriosclerosis has involved the artificial production of arteriosclerosis chiefly by the introduction of a number of chem-

ical and biologic substances into the circulation of animals. Many of these substances are those which have theoretically been suspected to be the direct exciting causes of arteriosclerosis or which have been demonstrated to be present in excess in such cases.

Among the more important substances held to be responsible for this condition are: the salts of cholin; excess of adrenalin in the circulating blood; toxins, as those of syphilis; acute infections; typhoid fever; etc. Poisonous substances resulting from abnormal proteid metabolism, as indol, amino-acids; excess of normal proteid material in the blood, and excess of cholesterin in the blood¹ (favored by excessive exercise, narcotics, adrenalin, etc.).

Among the many obstacles to the full understanding and satisfactory demonstration of the true relation of the exciting causes are the diversity of substances at present held to be responsible for the condition and which frequently may be coincidentally important. It is also a fact that all cases of arteriosclerosis do not present high blood-pressure, neither do all cases of high blood-pressure show evidences of arteriosclerosis; it is equally true that a large group of apparently dissimilar causes may result in arteriosclerosis: yet, nevertheless, the bulk of dependable evidence now tends to show that high blood-pressure usually precedes the permanent change in the arterial wall (see page 244).

S. E. DeSajous² attempts to overcome this apparent discrepancy by showing a logical biochemical relation between the majority of these agents. Thus he says: "While the

¹ Wacker and Houck, *Munch. Med. Wochen.*, Sept. 14, 1913.

² *Jour. A. M. A.*, Aug. 3, 1912, lix, 5.

adrenal secretion acts on the muscular fibers of the heart, and of the arteries in general, it has been found that the brunt of this action is borne by the arterioles. When such action is abnormal, we may have the production of arteriosclerosis. All the main causes usually assigned to arteriosclerosis may be summed up by the word 'intoxication.' All but one of the morbid conditions known to provoke arteriosclerosis (alcohol has not been studied) have also been shown to cause over-activity of the adrenals, and the evidence of this effect is further substantiated by the fact that Coplin, in an examination of the adrenals of twenty-two cases of arteriosclerosis, found that seventeen were markedly altered, the glands in the other cases being the seat of either tuberculosis or a secondary neoplasm."

Thayer and Brush¹ have studied at varying periods of months and years after their infection, 189 cases who passed through typhoid fever. Their results showed that between the ages of ten and fifty, 48.3 per cent. of old typhoids had palpable radials as compared with 17.5 among ordinary healthy individuals in whom serious infections and alcoholic habits had been excluded.

The average systolic pressure was materially higher for every decade in old typhoids as compared with normal.

The average size of the heart was larger among old typhoids than in normals when arranged in groups according to age.

They were also greatly impressed with the frequency of signs of endarteritis in the aorta and coronaries of patients dying with typhoid; this condition being found in fifty-

¹ W. S. Thayer and W. E. Brush, Jr., *Jour. A. M. A.*, Sept. 10, 1914, xliii, 11.

two out of ninety-five necropsies, while there was fresh arteriosclerosis in twenty-one instances.

They were unable to form any conclusive opinion in regard to the relation of venereal diseases because of the absurdly small proportion of cases who admit such infection.

In further substantiation of the infectious toxic theory, H. Huchard¹ reports an analysis of the etiology of 1835 cases of arteriosclerosis in the order of their frequency as follows: (1) rheumatism, 18 per cent.; (2) gout, 17 per cent.; (3) syphilis, 11 per cent.; (4) tobacco, 9 per cent.; (5) other acute toxemias, 3 per cent.; (6) diabetes, 2 per cent.; (7) alcohol, malaria, nervous causes, 1 per cent. or less; (8) unassignable causes, 30 per cent.

S. Dratschinski,² in a communication issuing from Metchnikoff's laboratory, confirms his previous announcement as to the toxicity of indol and its relation to the production of arteriosclerosis.

On the other hand the early work of Adler and Hensel³ obtained possible results of only twelve out of ninety rabbits in which they endeavored to produce arteriosclerosis by the injection of irritating drugs normally present in the human economy.

Stuckey has endeavored to show that the feeding of abnormal amounts of albuminous foods may be productive of arteriosclerosis. In experimenting with rabbits he succeeded in producing a hypertrophy of the intima, which was chronic and persistent in nature, and which appeared to be identical with the change seen in arteriosclerosis in man.

¹ *Bulletin de l'Acad. de Medecine*, July 7, 1908, lxxii, 27.

² *Annal. de l'Institut. Pasteur*, June, 1912, xxvi, 6.

³ *Jour. A. M. A.*, June 1, 1907, xlviii, 22, p. 1896.

Martin Bishoff¹ failed to produce arteriosclerotic changes by the administration of graduated amounts of alcohol to rabbits.

C. Hirsch and O. Thorspoken² administered adrenalin intravenously to rabbits and obtained advanced changes in the intima so intense as to remind one of well-marked arteriosclerosis in the human.

Popielski's belief in the physiologic behavior of the salts of cholin in raising blood-pressure and in the production of arteriosclerosis has not been substantiated, since Mendall, Underhill and Renshaw³ have noted that cholin salts produce a characteristic transitory fall of pressure; neither do they believe that the "contamination" theory is probable, as properly prepared and preserved cholin salts are not readily decomposed.

As the common effect of circulating toxins is a hyperemia and stimulation of the adrenals, it is fair to suppose that this relation, as suggested by Sajous,⁴ is to some degree at least an ever present one.

In demonstration of the toxic theory Y. Manouelian⁵ injected rabbits and monkeys with filtrates of emulsions of staphylococci and produced arteriosclerotic changes in 86 per cent. of the rabbits and in five out of six monkeys.

I. Adler⁶ has raised the question as to whether the effect on the vessel walls is due to the actual substance suspected or to some intermediate substance. When injecting dogs both intravenously and subcutaneously with

¹ *Zeit. f. exp. Path. im Therap.*, No. 11, 1912.

² *Deutsch. arch. f. klin. Med.*, 1912, No. 107.

³ *Jour. Pharmacol. and Exper. Therap.*, July, 1912, iii, 6.

⁴ *Loc. cit.*

⁵ *Annal. de l'Institut. Pasteur*, xxvii, No. 1.

⁶ *Jour. A. M. A.*, June 6, 1914, lxii, 23.

epinephrin, nicotin, lead, diphtheria toxin and pure cultures of different species of bacteria, he obtained no effect, while in a second series of experiments with these same substances, when administered orally, he obtained positive effects.

L. F. Bishop¹ states that heart disease and hardening of the arteries are nine times out of ten due to disturbance in the chemistry of the body (of the adrenals, the thyroid, toxemias, etc.), particularly in relation to the intestines and the liver. According to this authority the most important cause of arteriosclerosis is amino-acid poisoning, by acids to which the individual tissues are sensitive. Intestinal putrefaction has a very important bearing. He believes that idiosyncrasy accounts for the susceptibility in certain individuals as compared with others.

The amino-acids, which are responsible for these conditions, are, in all probability, derived from the breaking down in the intestinal tract of nitrogenous foods derived from milk, eggs, fish, meat and soups. And here he expressed the view already published by Rudolph² who quotes Huchard by saying, that the second most important factor in the production of arteriosclerosis is the continued excretion of toxins by the kidney, these toxins coming from abnormal proteid metabolism in the digestive tract and also in the liver.

INCIDENCE OF ARTERIOSCLEROSIS

While this disease usually is looked upon as one of the second half of life and is encountered usually during de-

¹ *Jour. A. M. A.*, Mar. 15, 1913, lx, No. 11.

² *Brit. Med. Jour.*, Nov. 26, 1910.

clining years, it should not be forgotten that well-marked cases are often met before the age of thirty and an occasional apoplexy has been reported in the early twenties.

The development of arterial disease and its complications are attracting more and more attention, because reports show the condition to be on the increase. Insurance statistics, particularly, point to the fact that both the age and incidence (development of symptoms) and the percentage of cases encountered are advancing, the disease being met in earlier life and more frequently than even a decade ago.

This unfortunate prevalence seems attributable to the increased tension and the greater artificiality of the life led by the average business and professional man to-day. Statistics show that men are more frequently victims of arteriosclerosis than women.

ETIOLOGY OF ARTERIOSCLEROSIS

Having discussed the "exciting agents" of arteriosclerosis, we now come to a consideration of what may be conveniently called the "productive or provocative causes." By this is meant those conditions which clinically are looked upon as entering actively into the etiology of the disease and which are found pathologically to be favorable to the development of this disease by contributing to the production or liberation of the "exciting agents" which are themselves directly responsible for the arterial changes found in arteriosclerosis (see page 246) and which are characteristic of the disease.

In reviewing the relation of these "provocative agents"

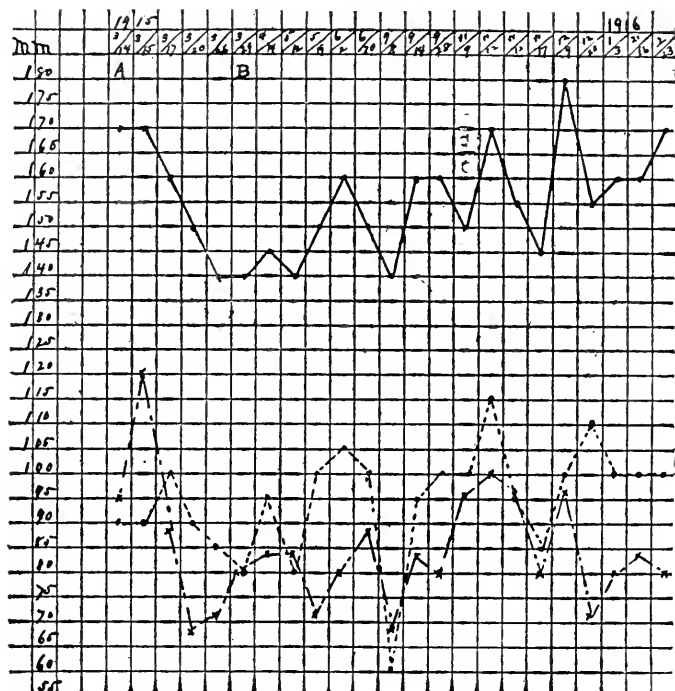


FIG. 49.—Female. Aged sixty-seven. Diagnosis: Chronic myocarditis and arteriosclerosis. Previous history one of hard work. Family history one of cardiovascular disease. First symptom dated four years previously and included loss of weight, dimness of vision, constant dyspnea with occasional orthopnea, palpitation, dizziness, headaches, edema of the ankles, physical weakness and abdominal pain. Examination shows moderate edema of legs up to knee, no ascites, a large and tender liver, transverse enlargement of the heart, chiefly to the left, apex not palpable, a slight systolic murmur at the apex, very irregular cardiac rhythm, including variations in strength of the impulses with intermissions and extra-systoles. Difference in 26 beats between radial count and apex count. Arteries slightly thickened, slight general cyanosis, urine shows large amount of albumen, normal specific gravity with hyaline casts not always present.

Morphine, rest and elimination resulted in reduction in pressure chiefly systolic from (A) to (B) accompanied by some relief from all symptoms. The dyspnea has been held in control by the intermittent alternating use of digitalis and strychnin.

The response to this treatment is gradually becoming lessened, while the tendency to an elevated systolic pressure becomes more persistent.

The case is one very difficult to manage and is working her own destruction by failure to adhere to restrictions particularly in regard to physical exertion.

Note. Since the preparation of this chart the patient has shown a systolic pressure persistently in the neighborhood of 200 with almost continuous orthopnea, nocturnal cardiac asthma and several attacks very suggestive of angina which are relieved by the use of heroin.

two difficulties are met in attempting to place formally the responsibility. First the indisputable fact that arteriosclerosis does not invariably follow in the wake of these causes, even when they appear to be very active and when their influence has persisted for a great length of time. Second, the almost universal existence of multiple "provocative causes" in the history of any given case, and the nearly utter impossibility of indicating definitely the single cause, if indeed any single cause be found to exist (see Fig. 49).

Accepting the existence of at times unsurmountable obstacles, in the way of isolating the cause of the disease in any particular case, we are now in a position to review the statistics available and to understand the apparent discrepancies found. Finally, to form our own conclusions, which can be adopted to our individual needs and employed as diagnostic leads in the course of practice.

One of the earliest statistical reports is that of Huchard¹ who reported the causative factor in 2680 cases of arteriosclerosis found in 15,000 patients seen in the course of private practice. In their relative frequency these were as follows: gout, uricemia, food, syphilis, tobacco poisoning, worry, mental over-exertion and alcohol.

Herz's² more extensive study covered the tabulation of reports of the most frequent cause furnished him by 822 different observers. This composite report gives as follows the relative frequency of causes as met by each contributor:

¹ *Med. klin. Berlin*, v, No. 35.

² *Wien. klin. Wochen.*, xxiv, No. 44.

Emotional and nervous.....	150
Physical exertion.....	146
Age.....	138
Alcohol.....	133
Tobacco.....	88
Syphilis.....	77
Heredity.....	72
Metabolic disturbances.....	19
Coffee and tea.....	13
Infections, etc.....	7

Thayer¹ tabulates his study of 3894 cases in their order of frequency as follows: heavy work (62.2 per cent.), alcohol, rheumatism, diphtheria, scarlet fever, malaria and pneumonia.

Brooks² studied 400 cases and heads the list with occupational lead poisoning, severe manual labor, alcohol, nephritis, syphilis and tuberculosis.

Thayer and Brush admit that the majority of cases give a history of multiple factors which they do not attempt to separate but which they divide into four groups:

First.—Manual labor, 57.6 per cent.

Second.—Alcohol, 46.8 per cent.

Third.—Acute infections.

(a) Rheumatism, 34.6 per cent.

(b) Typhoid fever, 26.3 per cent.

Fourth.—Those having acute infections alone (variety not stated), 24.3 per cent.

Rodger I. Lee³ reports the following causes present in the percentages given below which in the majority of instances were multiple.

¹ *N. Y. Med. Jour.*, June 18, 1904.

² *A. M. A. Jour. Med. Sci.*, May, 1906.

³ *Jour. A. M. A.*, Oct. 7, 1911, lvii, No. 15.

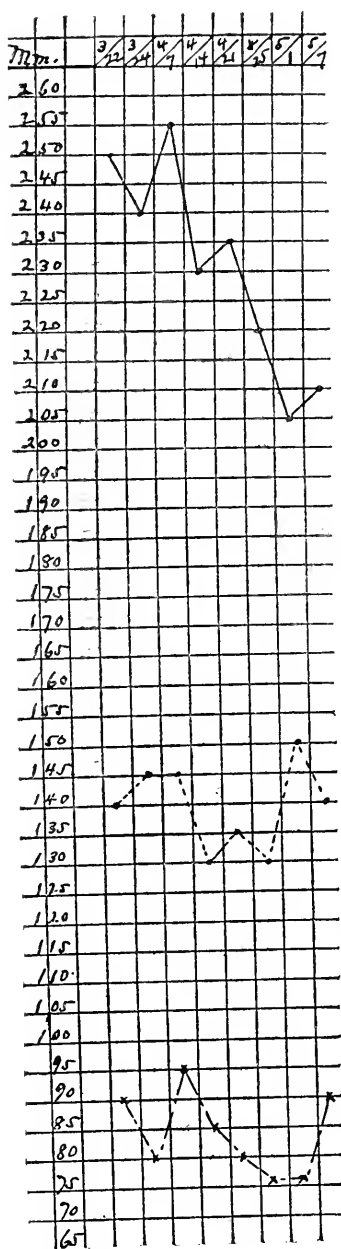


Fig. 50.—Female. Aged fifty-four. Post-uremic. Arteriosclerosis and contracted kidney. Albuminuric retinitis first called attention to this condition three or four years ago. Chief complaint nervousness, depressive dreams, and hallucinations of oppression and fear. Is inclined to weep and looks upon the dark side of things. Complaints of poor vision with buzzing and roaring in ears, physical weakness and occasional attacks of nausea, pain in the head, nocturia and occasional dyspnea.

Examination shows elongated and fibrous arteries, slight cyanosis, large liver, heart enlarged 1 in. to the right and an accentuated aortic second sound with weak muscle sounds. Urinalysis shows albumen and occasional hyaline, granular and fatty casts. Blood cells are continuously present.

The high systolic pressure and the very large pulse pressure indicating a marked arteriosclerotic involvement. Treatment in this case was almost wholly dietetic and hygienic with routine hot-vapor baths. Small doses of iodides in various form were tried but on account of increase in heat flashes and head symptoms were discontinued. The vapor baths in this case averaged three a week, sweating began in three minutes and was continued for ten. There was no after exhaustion or any untoward results. The head relief was marked. The phthalein test made 3-24-'15 showed 50 per cent. first hour, 25 per cent. second hour, total 75 per cent.

	Per cent.
Kidney lesions.....	71
Arteriosclerosis.....	69
Arteriosclerosis and kidney combined.....	52
Cerebral lesions.....	15
Cardiac lesions.....	24
High pressure and arteriosclerosis (kidney normal).....	28
Pure hypertension.....	13

Janeway has been led to believe that, in cases of generalized arteriosclerosis which show high blood-pressure (Fig. 50) this pressure is largely due to a kidney condition as most cases of generalized arteriosclerosis without kidney involvement are unassociated with high pressure (about 50 per cent.).

This observer was fortunate in being able to follow many of his cases to necropsy, thereby being able to compare the clinical and pathologic diagnosis. His study brought out the marked imperfections in our present methods of diagnosis, by demonstrating that a large percentage of clinical diagnoses were either incorrect or incomplete when checked by the pathologic report.

SUMMARY

Considering all the evidence, a few important facts stand out clearly, namely, (a) that well-developed generalized arteriosclerosis affects blood-pressure chiefly when the arteries of the general arterial tree are involved, without which blood-pressure may not be altered even by marked local arteriosclerosis. (b) That thickening and degenerative changes in the intima result wholly or in part through the irritating effect of abnormal metabolites or other irritating substances circulating in the blood, and that these substances may operate through the agency of the nervous

system through stimulation and congestion of the adrenals or directly by local irritation upon the vessel lining. (c) The origin of these substances is diverse, probably in the majority of cases of gastro-intestinal or liver origin, although toxic substances of an infectious nature are also of great importance. (d) The majority of cases of arteriosclerosis are preceded by a period of slightly elevated blood-pressure due to a narrowing or hypertension of the arterial walls; and this change is at first transitory, but by being constantly repeated the arterial system is subjected to an intermittent trauma, which is productive of a permanent alteration of a degenerative character in the fibrous coats of the arteries. (e) This change is largely a protective effort on the part of nature to strengthen the artery in order to withstand the added strain. In order to overcome the resistance offered by the hypertension (f) the heart hypertrophies, while disturbance in renal circulation interferes with proper elimination, thus throwing additional irritating substances into the circulation, thereby establishing a vicious circle, which gradually increases as the pressure mounts higher and higher, unless relief comes from a break at some point.

PATHOLOGY

The term arteriosclerosis is too loosely employed by the average physician. This has led to great confusion in the reporting of cases and in the compilation of statistics. It is often impossible to learn precisely what condition an author is discussing, so that the benefits of careful research are often lost to the reader. The two conditions usually confused are, atheroma and diffuse generalized arterio-

sclerosis, and less often the condition of pure hypertension, as found before any permanent change has occurred in the vessel wall (see page 244).

The pathologist has more than once pointed out clearly these different conditions and has correlated them with the physical signs. Among them Russell has made most careful studies of the condition of the vessels, and their relation to chronic disease of the heart, kidneys, cerebral system, and to blood-pressure. According to Russell,¹ atheroma is a local or patchy affection of the arteries characterized by a local thickening and degeneration of the intima. This soon undergoes a form of fatty degeneration which is termed, atheroma. Later these patches become the seat of a calcareous deposit while in the larger arteries atheromatous cysts and ulcers may be formed with local sacculations. These changes may be so extensive, especially in the aortic arch that a local bulging occurs to which the name aneurysmal bulging has been applied.

Atheromatous changes are quite common in the cerebral and coronary arteries but comparatively rare in the radials. When present in the radials, they give rise to local thickenings, which give an irregular nodular feel to the vessel. They are never symmetric. Russell believes that the character of these changes is very suggestive of a low-grade infection, and assigns a primary micro-organismal implantation as their origin.

Arteriosclerosis, on the other hand, may be roughly defined as a thickening of the arterial wall with a diminution in the size of its lumen. The changes which have led

¹ Wm. Russell, "Arterial Hypertension, Sclerosis and Blood-pressure," J. B. Lippincott Co., 1910.

to this when examined in detail are seen to consist of (1) a marked thickening of the intima, due to hypertrophy of the muscle fibers; (2) a thickening of the intima without atheromatous degeneration; (3) and in some cases a fibrous thickening of the adventitia. The muscular coat may show some degeneration but the prevailing notion that in such thickened vessels the muscle coat is replaced by fibrous tissue (fibrous degeneration) is erroneous (Russell) (Thayer and Brush).

These changes are not confined to limited areas of the vessel wall as in atheroma, but affect uniformly a large portion of the vascular system and are usually distributed throughout the body, for instance, in the coronary and renal arteries.

Cases are encountered where both processes are met in combination. Usually these occur late in life, the atheromatous changes generally being confined to the large vessels and aorta.

The clinical study of blood-pressure and its relation to visceral involvement would seem to bear witness to the accuracy of Russell's deductions and conclusions, for it will be recognized that were this change one of pure fibrous degeneration with destruction of the muscular tissue in the vessel walls, then measures directed toward relieving hypertension (contraction of the muscular wall) would be useless. As proof of this and of the value of such measures, we have only to review the evidence found in every-day practice, where such measures effect a reduction in a large majority of cases.

CLINICAL MANIFESTATIONS

Clinically the elevation of pressure in arteriosclerosis affords a method of distinguishing between this disease

and atheroma with which it is so often confused. Atheroma is really a senile affection, coming on in persons between sixty and eighty years and which involves the blood-vessels only. Arteriosclerosis on the other hand may attack persons between thirty and sixty years of age and is largely a visceral complaint involving as it progresses, the heart, kidneys and nervous system. Although Oppenheim¹ has reported two cases of undoubted arteriosclerosis in boys of nine and ten years of age. The first died of spontaneous rupture of the aorta, probably of syphilitic origin, while the second case was undoubtedly due to autotoxemia.

With the study of atheroma we are but little concerned as this condition must be looked upon as a more or less natural process due to the changes caused by advancing years, and not particularly related to those factors which are recognized as producing arteriosclerosis.

Gull and Sutton's original conception of this disease as an "Arterio-capillary Fibrosis" is incomplete. From the viewpoint of the pathologist, the clinician and the therapist, we must recognize the multiplicity of the causes involved in arteriosclerosis and admit that the condition is usually a joint involvement of the heart, the blood-vessels and the kidneys, in which the arterial changes may be determined to be the predominant factor in a certain per cent. of cases.

From a purely clinical standpoint it would seem convenient, even if not scientifically correct, to divide the evolution of arteriosclerosis according to the four divisions first suggested by Huchard.² Thus:

¹ *Virch. Arch.*, vol. clviii, No. 2.

² *Jour. A. M. A.*, Vol. liii, No. 14, p. 1129.

1. Arterial hypertension (presclerosis).
2. The cardioarterial stage.
3. The reno-arterial stage.
4. The final stage of cardiac failure.

Such a classification will be found very valuable to the practising physician, because the mere placing of a case in one of these divisions (and it is usually possible to do this) will at once greatly aid in giving a prognosis, and also suggest the character of the treatment indicated.

Clinically, arteriosclerosis may begin in the kidneys, in the heart, in the extremities (in laborers or farmers), in the brain, or in the mesentery; but there cannot be generalized arteriosclerosis without both cardiac and renal involvement.

SYMPTOMATOLOGY

In the cases presenting pipe-stem and tortuous arteries with ringing aortic second-sound and a long list of suggestive cardiorenal symptoms, so often seen in elderly individuals with interstitial nephritis, the diagnosis is made for us, and the treatment is of little avail. It is to the early and unsuspected cases, those presenting few if any symptoms or signs of disease, in which there is present little or no palpable change in the peripheral arteries, doubtful change in the valve sounds, perhaps a little roughening of the second sound, with a normal or practically normal urine, that our attention should be directed. These cases may and usually do, after diligent search, show obscure gastro-intestinal symptoms which so frequently fail to obtain proper consideration from the clinician. Such cases usually have a systolic range of from 160 to 250 mm. Hg. (see Fig. 53), and an increased pulse pressure of which the increase in

pulse pressure is of greatest significance. For this will be found in arteriosclerosis even when accompanied by a normal systolic pressure.

In persons about to enter or already in middle life, these blood-pressure changes, in the absence of demonstrable nephritis, may be the only physical sign pointing directly toward the diagnosis.

With regard to an exact symptomatology of early generalized arteriosclerosis, the clinical signs and subjective symptoms may simulate almost any known disease and present almost any clinical signs, from a fleeting dizziness to gangrene of the extremities. Some of the symptoms are not infrequently attributed to neurasthenia. There are vague, unpleasant feelings or fullness in the head, slight momentary dizziness, cold hands and cold feet, sleep unrefreshing and disturbed by dreams, gastric distress and flatulence coming on one or two hours after meals, constipation and loss of power of concentration and interest in business affairs. The general vitality and power of resistance of the body is less than formerly and tolerance for substances which affect the brain, as alcohol and tobacco is often diminished. The patients tire easily. As the process advances the gastric symptoms increase in severity and exertion after meals may bring on attacks of gastric and heart pain, which are relieved only by resting. Such individuals become incapacitated for work. They are nervous, lose weight and move slowly. Evidence of involvement of all the organs in the arteriosclerotic process appears, notably in the brain, the heart, the eyes and the kidneys. Extreme cases give all the classical symptoms and signs which go to make up the syndrome of cardio-

vascular-renal disease. By this time the diagnosis is as easy as the treatment is difficult.

Cases of arteriosclerosis which have sustained a high

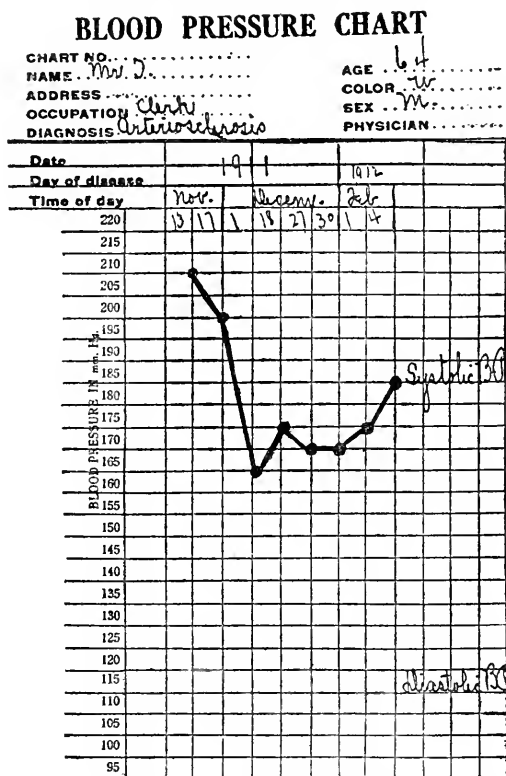


FIG. 51.—Case sought advice because of the recurrence of an old sciatica. Treatment was largely directed toward a chronic intestinal toxemia. The marked fall in pressure resulting from a relaxation of hypertonus, while the further failure to bring the pressure below 165 indicates the failure of such measures to affect a permanent change in the vessel walls.

pressure over considerable time show periods of great depression with severe headaches, nausea and sudden vertigo. This is due to the irritation and diminished nutrition of the cerebral centers from the high pressure and the narrowed

arteries. Eventually these symptoms become more or less constant, memory fails and insomnia ensues, while life becomes a burden.¹

BLOOD PRESSURE CHART

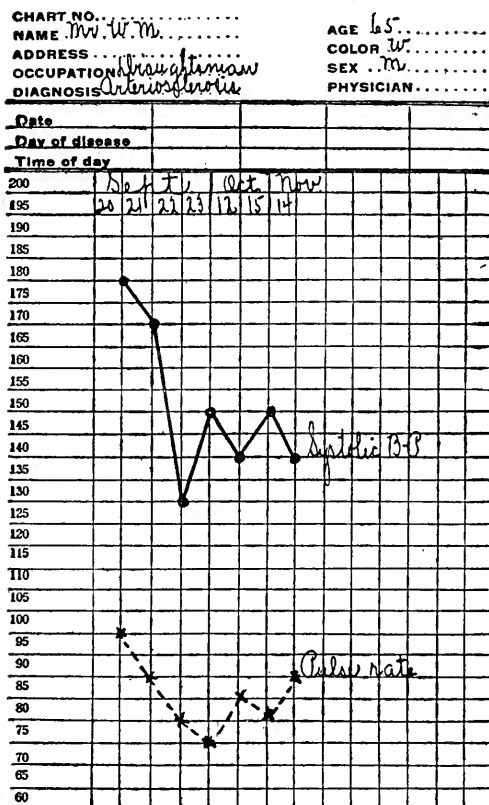


FIG. 52.—Symptoms chiefly cerebral, September 20, patient slightly delirious, some muscular weakness on left side of body. Chart shows effect of active treatment continued until September 23. Remainder of chart shows lowered pressure maintained by physical measures after patient resumed his activity.

Often it is not until these cases suffer a cerebral hemor-

¹ Cerebral type of Bing, R. Bing, *Correspond. bl. d. Schw. Aerz.*, August, 1912, xlii, 22.

rhage or show signs of cardiac weakness, that they are even suspected of having anything more than a nervous condition. Routine observations of blood-pressure and a practical knowledge of the early signs of this disease are essential if a diagnosis is to be made in time to obtain benefit from preventive measures. The routine estimation of blood-pressure and the preparation of a daily chart is of great value in the study of suspicious cases such as simple chronic bronchitis with emphysema, which are often explained and their etiologic factors supplied by demonstrating the presence of continued high pressure. Indeed the blood-pressure need not be very much elevated to injure the heart and other organs, for an increase of a few millimeters, if long continued, entails an enormous increase in the daily work of the heart.

DIAGNOSIS

Of primary importance in the diagnosis of any chronic condition is the taking of a thorough, carefully arranged and comprehensive history. It should cover all points in the patient's personal, social and previous medical history. Take nothing for granted and leave no important point uninvestigated. Properly taken and carefully summarized the history alone will often suggest the diagnosis and will indicate the direction for special study which may serve to establish a definite diagnosis.

The preceding paragraphs have, I think, sufficiently emphasized the important symptoms which should be sought so that repetition is unnecessary. A thorough physical examination is desirable, followed by such subsequent special examinations as are indicated by already

acquired information. These will usually include the blood-pressure and the functional capacity of the circulation by the means of the sphygmomanometer, the condition of the blood, of the eye-grounds and of the urine.

Examination of the Arteries.—For diagnostic purposes we may assume that the arterial wall may usually be demonstrated, by palpation, to be thickened after the age of forty years (Daland¹). This seems very practical from a diagnostic standpoint when we consider that a demonstration of the state of the vessels is purely a relative comparison and that to ignore this premise would lead into error resulting in a diagnosis of arteriosclerosis in patients having vessels with no more than a normal degree of thickening.

Observation is, therefore, first directed toward a study of all accessible vessels by means of inspection and palpation, not forgetting those of the retina by means of the ophthalmoscope. The study of the radial arteries gives most valuable information, but it must not be forgotten that the fibrotic process may be inconspicuous in the peripheral arteries while well advanced in the internal arteries, more especially the splanchnic and cerebrals; and that occasionally fibrosis may be advanced in the peripheral vessels but little or no change in the important internal ones. On account of variations in size and situation of the radial arteries both should be examined. It must also be remembered that the excessive deposition of adipose, or the presence of edema, may prevent successful examination of the radial arteries. It seldom happens, however, that radial sclerosis is diagnosed when absent, the error is usually on the other side.

¹Judson Daland, *Monthly Cycl. Pract. Med.*, Vol. x, p. 145, 1907.

It is important to separate true sclerosis from pure hypertension as the impression under the finger in these two conditions is quite similar. Arterial spasm usually occurs in the young and palpation of the vessel wall reveals a vessel which feels thicker and smaller than normal, while the lumen appears to be diminished. The common causes of this condition are (1) acute uremia, occurring in the course of acute parenchymatous nephritis, as in scarlet fever and similar infectious processes; (2) in certain cases of severe acute intestinal toxemia; (3) in certain cases of irritating chemical poisoning.

Apart from the result obtained from palpation, the cardiac and renal signs of arterial spasm may exactly simulate arteriosclerosis and we are therefore compelled to rely upon the knowledge of the cause and duration of the condition to determine the degree of arterial change.

Reliance should not be placed upon the radial arteries alone, but for diagnostic purposes we should use the temporal, the carotid, the brachial, the abdominal aorta, the femoral and the dorsalis pedis.

Changes in Blood-pressure.—Having determined the condition of the blood-vessels, blood-pressure tests may then be applied. In the presence of arteriosclerosis the systolic pressure will be found above that determined as normal for the age of the individual. This elevation need not be great. A continued hypertension of 20 or 30 mm. unless explained upon other grounds should be considered pathologic and call for explanation. It should, however, be remembered that cases will be met having very hard and firm peripheral vessels yet which have a normal or subnormal systolic blood-pressure.

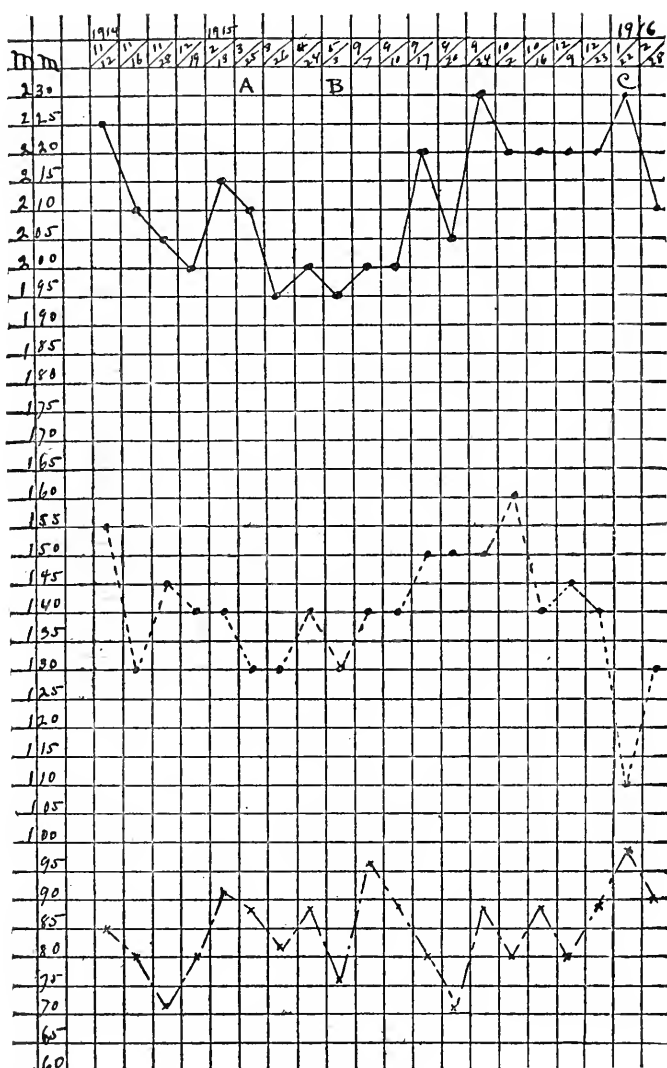


FIG. 53.—Arteriosclerosis. Aged forty-three. Clerk. Complains of persistent headaches for a period of about three years, not relieved by glasses. Prior to the first observation, he is said to have had a pressure of about 200. Attempts to reduce this by appropriate electrical treatment were unsuccessful. The headache is characteristically one of arteriosclerosis, coming on in the early morning, usually waking the patient between 5 and 6, and wearing

I distinctly remember one case of over fifty years of age having the most rigid and pipe-stem radials that I have ever palpated, yet at no time was the systolic pressure found to be over 100 mm. Hg. Again the hypertensive effect of arteriosclerosis may be counteracted by the hypotensive effect of an associated thyroid disease or a deranged adrenal system, as in Addison's disease (see page 216).

A comparison of the systolic and diastolic pressures and an estimation of the pulse pressure is of distinct value in the study of all cases. The physical changes produced in the dynamics of the circulation by the less elastic vessel walls, will in the presence of a normal heart show an increased pressure, often as high as 60 and occasionally 100 or more, as the advanced and extreme cases are met (see Fig. 53).

Examination of the heart in pure early arteriosclerosis (before the kidney has become much damaged) will reveal

off during the day. Is usually left-sided extending from behind the ear to the frontal region and involving the eyes.

Has been an excessive alcohol user, but has during the past several years totally abstained.

Dietetic and hygienic treatment accomplished much in the relief of the headache without greatly affecting blood-pressure. The drug medication was entirely incidental to a condition of the stomach, bowels and intercurrent colds. The phthalein test on 9-24-'15 gave a 50 per cent. output in the first two hours. The urine has occasionally shown a trace of albumin, has been of normal specific gravity and has variably shown casts both hyaline and granular and a few blood cells.

The depression in systolic pressure between (*A*) and (*B*) was incident to a development of an acute mastoiditis necessitating confinement to bed. At (*C*) the patient presented with signs of pulmonary edema which was promptly relieved by appropriate treatment, coincident with a reduction in both systolic and pulse pressure.

From a survey of this chart it is evident that the condition contributed to the high pressure is progressing in spite of measures directed toward its relief and it is only a question of time, possibly short, when this patient will succumb to the effects of an ever-increasing systolic and pulse pressure.

some slight accentuation of the second aortic sound, and a compensatory hypertrophy, although the studies of Romberg and Hasenfeld¹ found hypertrophy of the left ventricle in only a small proportion of cases of arteriosclerosis before an associated nephritis had developed.

The temperature, as is the case in most chronic diseases, will usually be found subnormal, although Stengel² calls attention to the occurrence of continued fever in certain cases, and he holds that when there is no other assignable cause for the fever, it is probably due to the arteriosclerotic process.

The Digestive Tract.—Examination of the digestive tract will often reveal slight departures from normal often dating back for many years. The results of test-meal examinations will show reduced gastric secretory activity. There is usually abdominal distention and often most obstinate constipation.

The *ocular changes* found in arteriosclerosis are corkscrew retinal artery twigs, a dull red nerve head and flattening of the veins at the arterial crossings. Sluggish pupillary reaction and early failure of accommodation.

Pathognomonic evidence of arteriosclerosis will be found by the presence of most or all of the following ocular signs:

1. Change in the course and size and irregular caliber of the arteries and of the veins, the latter being indented at the arterial crossings.
2. Altered vascular reflexes, such as more brilliant central light streak (silver wire artery).
3. Undue fullness of the perivascular lymph-sheaths.

¹ *Deut. Arch. f. klin. Med.*, Vol. lix, 1897, p. 193.

² *Medicine*, Detroit, June, 1906.

4. Paleness of the vessels.

In more advanced cases;

5. Faint hemorrhages.

6. Retinal haze, more marked at the nerve head.

The "red-hot" eye is frequently seen in arteriosclerosis particularly in the presence of a gouty diathesis. A persistent asthenopia and atrophy are also encountered.

Hertzell's¹ *reaction of congestion* for the diagnosis of arteriosclerosis is only mentioned to be condemned, for any procedure which contemplates complete interruption of the circulation in the extremities with a consequent rise of 60 or more millimeters of mercury in an arteriosclerotic, is to say the least hazardous in the extreme.

¹ C. Hertzell, *Berl. klin. Wochen.*, Mar. 24, 1913.

CHAPTER XVIII

NEPHRITIC HYPERTENSION AND CHRONIC NEPHRITIS

Etiology.—The question of the etiology of the several distinct types of chronic Bright's disease such as chronic parenchymatous nephritis, chronic interstitial nephritis and syphilitic nephritis is still under discussion. A careful review of the data at hand shows that the last word has not yet been said, neither has it been settled whether the arteriovascular changes are primary or secondary to the renal changes. There being much equally reliable evidence pointing in both directions, it would seem expedient to assume a middle position and admit for the present, at least, that in view of the various and diversified causes of arteriosclerosis and nephritis, it is probable that the arterial process may be primary in some cases, the nephritic involvement the first pathologic change in others, while in a third group the two may run parallel courses. This position is strengthened by the undoubted fact that the majority of post-mortem reports, as well as the bulk of experimental evidence, tend to show that either condition may exist independently of the other, although usually they are found combined.

A dissenting opinion is that of McCrae¹ who believes that,

¹ McCrae in Osler's "Modern Medicine," 1909, vi.

neither chronic parenchymatous nephritis nor chronic interstitial nephritis ever exists alone, in as much as "interstitial change never exists without an accompanying parenchymatous change and parenchymatous change cannot exist long without some interstitial alteration following or accompanying it." He doubts the development of a primary contracted kidney.

Strumpell considers chronic interstitial nephritis as an atrophy of the parenchyma which begins in a previously healthy kidney, and not necessarily the ultimate termination of a chronic diffuse nephritis, though he describes a secondary contracted kidney which he considers simply a more advanced form of chronic diffuse nephritis.

Admitting the possibility of an occasional pure case of either type, we are forced to admit, since both morbid anatomists and pathologists are far from unanimous in their descriptions of the various types and groups of chronic nephritis and as physicians are not always able to make a differentiation that is satisfactory from a clinical point of view or that holds good in the light of post-mortem revelations, that by far the largest number of cases met in actual practice belong to what Dieulafoy¹ calls mixed nephritis and that these constitute the common form of chronic Bright's disease.

Concerning the relationship of arteriosclerosis to chronic Bright's disease, we are just as much in the dark, because, if we accept the general theory that the lesions of chronic contracted kidney are due to a local form of arteriosclerosis, this does not explain all the cases of chronic nephritis in which the arteries chiefly are affected, nor does it fit in

¹ "Text-book of Medicine," 1911, ii.

with the fact that we may have extensive arteriosclerotic changes with very little renal change.

In the light of this continued uncertainty it would seem best, from the view point of a clinician, to subordinate these degenerative lesions, not only in the kidneys but also in other organs commonly affected, to the one prime or provocative cause (as gout, lead infections, heredity, etc.) (see page 261).

It is also a well-known clinical fact that it is rare to find a case of chronic interstitial nephritis, which permits of a fairly definite diagnosis, in which the cardiovascular changes are not more or less well developed.

We have then, in the average case, a pathologic condition which involves jointly, chronic changes in the arteries, in the kidneys and in the heart; in the study of which our diagnostic effort is directed, first, toward placing the chief responsibility and second, toward arresting the process or discovering and rendering inactive the cause or causes. The solution of this problem is not simple, neither is it always possible, so that we may be forced to be content with the general diagnosis of cardiovascular renal disease; because it is now accepted that (a) arteriosclerosis may cause the arteriosclerotic kidney, (b) contracted kidney may cause arteriosclerosis, or (c) both may result from a common cause. However this may be, it is evident that the provocative substance reaches the kidneys through the blood-stream, which fact has probably led Anders and others to consider chronic interstitial nephritis as but one lesion of a generalized process of fibrosis, in which no one can predetermine what organ or organs will first succumb to the toxic irritant.

Despite many opinions to the contrary, it would seem reasonable to believe that no matter what the cause may be, hypertension is the primary trouble, that this results in a dilatation of the arterial walls, incident to the increase in lateral pressure; while nature in an effort to combat the abnormal dilatation produces a gradual thickening in the arterial wall which is the first step in the production of arteriosclerosis. Either coincidentally or immediately following this, the increased pressure and the change in the arterial walls demand additional work of the heart, to provide which the heart hypertrophies, thereby setting up a portion of a vicious circle which is completed when the arteries and arterioles of the kidneys take part in the degenerative process which leads to an arteriosclerotic, or chronically contracted kidney. This theory is further strengthened by the fact that it is reasonable to suppose, and has been experimentally demonstrated, that the toxins which are directly responsible for the rise in blood-pressure at the same time exert an injurious effect upon the arterial walls and that, where the action of these toxins is long continued, the persistent hypertension becomes a permanent high blood-pressure, with the development of general arteriocapillary sclerosis, as was first suggested by Gull and Sutton in 1872.¹ Again, it is now generally believed that no marked general arteriosclerotic changes can exist without more or less involvement of the kidneys. Whether the renal lesion will be the predominating factor depends entirely on the original integrity of the kidneys, and their power to resist the effect of the toxin which primarily caused the vascular change.

¹ *Med. Chir. Trans.*, 1872, lv.

THEORY OF THE MECHANISM OF THE PRODUCTION OF HIGH BLOOD-PRESSURE

Several theories have been advanced to explain the relation of the kidney to the high blood-pressure in nephritis, none of which have been fully accepted as conclusive.

We may conveniently divide these theories into:

- (a) The mechanical.
- (b) The chemical.
- (c) The retentive.

(a) **Mechanical Theory.**—There are still many supporters of the old Traube and Cohnheim theory, which held that mechanical interference with the blood flow in the kidney is responsible for the high blood-pressure in nephritis. Failure to produce increased blood-pressure by ligating the renal arteries or by the experimental production of multiple renal emboli does not disprove this theory. It has been suggested that the increase in blood-pressure is a protective effort on the part of nature to promote an adequate kidney function in the face of mechanical deficiency resulting from glomerular destruction. This theory is hardly tenable in the face of the fact that in amyloid disease of the kidneys (a glomerular affection) the blood-pressure is rarely affected and the heart not often hypertrophied.

(b) **Chemical Theory.**—This theory is complicated, in that it attributes the high blood-pressure to the activity of chemical substances derived from several sources resulting in (1) a reduced or altered renal elimination, (2) a disturbance of internal secretion, (3) toxic substances liberated by the diseased kidneys.

1. In an effort to prove the reduced elimination theory

Pearce¹ found that extracts of the kidneys of dogs, having experimental uranium or chromium nephritis, when the urine showed none of the normally present depressor substance, had no pressor effect, and that it is therefore improbable that the kidneys are responsible for a pressor substance of importance in the pathology of cardiovascular and renal disturbances.

2. The hyperperformance of the adrenals has long been advanced as the probable cause of a continued high pressure. Recent investigations, however, have failed to demonstrate, in the patients with hypertension, any increase in the epinephrin in the circulating blood. Furthermore, R. G. Hoskins and C. W. McClure² state that the primary affect of adrenalin administered intravenously is a lowering of blood-pressure and that the quantity required to cause (experimentally) a minimum hypertension is several times the quantity that they were able to demonstrate experimentally in the circulating blood.

Recently Voegtlin and Macht³ after reviewing the evidence for and against the direct relation of epinephrin in the blood-stream to the blood-pressure level, agree with O'Connor⁴ that there is some other vasoconstrictor substance in the blood. This they believe to be the crystalline substance isolated and described by Zucker and Stewart⁵ and by Kaufman.⁶ They have been able to isolate the substance already described by these observers and to make

¹ *Arch. Int. Med.*, May, 1912.

² *Arch. Int. Med.*, October, 1912, x, 4.

³ Carl Voegtlin and David I. Macht, *Jour. A. M. A.*, Dec. 13, 1913, Vol. xli, 24, p. 2136.

⁴ *Arch. f. exper. Path. u. Pharmacol.*, 1911, lxiii, 195.

⁵ *Jour. Exp. Med.*, 1913, xvii, 152.

⁶ *Centralbl. f. Physiol.*, 1913, xxvii, 5270.

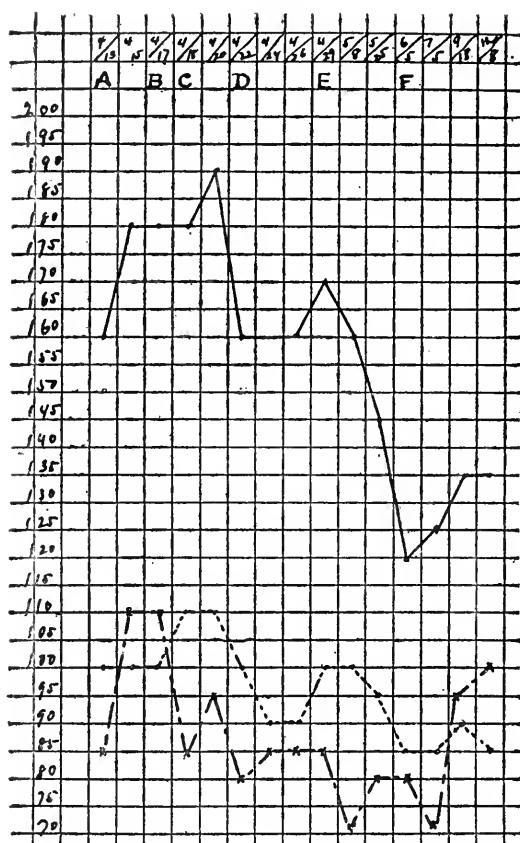


FIG. 54.—Male. Aged thirty-nine. Acute toxic irritation of kidneys. This patient has been personally known to the author for fifteen years and has enjoyed particularly good health.

At (A) examined and at that time stated that he had not felt well for about two weeks and dated his trouble from an attack of food poisoning with moderate prostration and diarrhea. This was followed by a sore throat which developed into tonsillitis. He complained of feeling tired and weak, although he continued work. Later his hands became swollen, his shoes tight and a persistent headache developed which was intense. Nausea was persistent and during the preceding twelve hours all food had been vomited. Examination showed an abnormally high systolic pressure and large pulse pressure, an irregular pulse, an accentuated aortic second sound, a normal temperature, scanty urine of high specific gravity, a large amount of albumen, an occasional hyaline cast and a few red blood cells. In spite of restricted

a study of its chemical, physical and physiologic properties, from which they feel justified in regarding it as a definite chemical substance, with a pharmacologic action different from epinephrin, and from any other body hitherto obtained from the blood. Considerable importance attaches to this discovery, as it appears that this substance may be the long-sought cause of high blood-pressure in cardiovascular-renal disease.

M. Hagelberg¹ found a high sugar content of the blood in twenty-six cases which he studied. He suggests that this condition may be the cause of the disease and that it may indirectly be due to the supposed increase in epinephrin content in the blood, whereas A. Farini² found no relation between the amount of excess of sugar in the circulating blood and the presence of arteriosclerosis or chronic nephritis.

3. One of the most important factors in the production

diet and of eliminative measures the pressure continued to rise for a week, although the headache and nausea were promptly relieved at (B).

The phthalein output at (C) was: first hour 47 per cent., second hour 36 per cent., while the urinary output had greatly increased and the albumen was reduced.

At (D) the patient expressed himself as feeling perfectly well but was still confined to bed on account of evidence of cardiac over-action as shown by the pulse pressure. At this time the urine had returned to a normal specific gravity, the albumen was reduced to a trace and there were still occasional hyaline casts and cylindroids, and an occasional red blood cell.

At (E) the patient resumed moderate activity and was symptom free.

At (F) he returned to business and the embargo on meat was removed and the patient dismissed.

Subsequently the slight rise in pressure is explained by increased physical activity, as there has been no evidence of the original symptoms while urine remains normal. The patient has reported recently that he is in excellent condition.

¹ *Berl. klin. Wochen.*, Sept. 20, 1912, xlix, 40.

² *Gazet. d. Ospedali e dec Cliniche*, August, 1913, 92, p. 951.

of arteriosclerosis is the continued excretion, by the kidneys, of toxins coming from abnormal proteid metabolism, in the digestive tract and in the liver (Fig. 54)—substances which have long been considered to be important factors in the production of contracted kidney and of arteriosclerosis. Indol has been recently shown by Dratschiniski¹ to be of great importance in this connection. Other experimental evidence has shown that to produce polyuria and increased blood-pressure by mechanical destruction of kidney structure, about two-thirds of the total kidney substance must be removed and that it is impossible to say whether these results are due to lessened elimination or mechanical interference with the renal blood-supply, and further, that while in uremia there is usually nitrogenous retention, it has not been shown that such retention is responsible for the increased blood-pressure.

(c) **Retention Theory.**—E. Krautenberg² demonstrated experimentally the production of a chronic nephritis in rabbits by ligating the ureter. Autopsy on such animals, often alive twelve to eighteen months after the ligation, always showed extensive sclerosis and aneurism of the aorta, which this observer attributes to blood-pressure changes resulting from the kidney degeneration caused by the ligation.

H. H. Mills³ advances the theory and cites cases in which he found marked diminution in the urea excretion in chronic nephritis as compared with a number of normal cases in which the pressure and kidneys appeared normal.

The attempt has been made to explain high blood-pres-

¹ *Annal. de l'Institut. Pasteur*, June, 1912, xxvi, 6.

² *Deutsch. Med. Wochen.*, March 25, xxxvi, 12.

³ *N. Y. Med. Jour.*, Aug. 20, 1910.

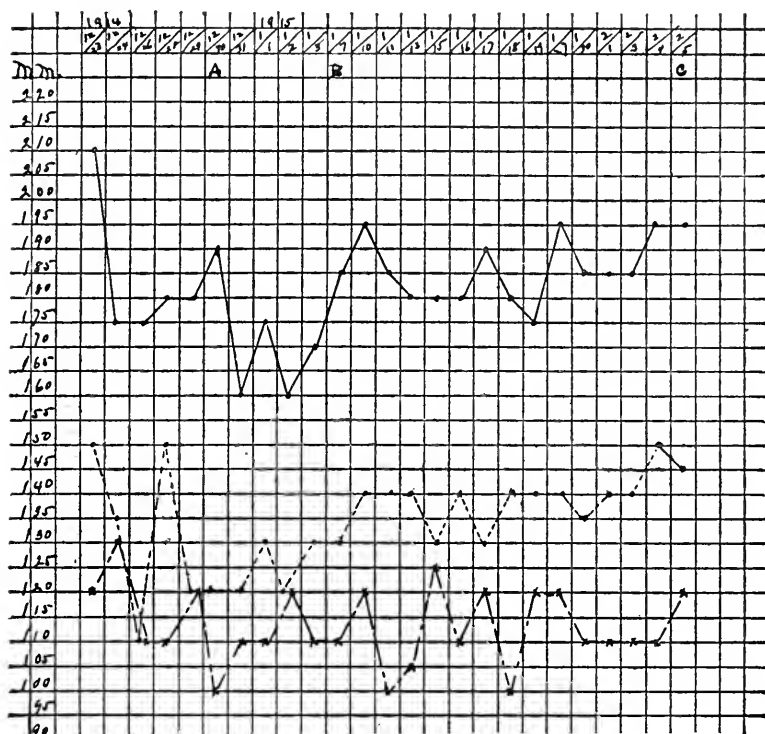


FIG. 55.—Acute parenchymatous nephritis. Male. Aged twenty-nine. The first evidence of this condition was the sudden development of almost complete blindness, due to extensive retinal hemorrhage as shown by ophthalmoscopic examination. Heart rapidly became over-active and dilated. Cardiac second-sounds very sharp and a systolic murmur developed. Urinalysis showed an immense amount of albumin, all varieties of casts and blood cells which persisted. Cardiac embarrassment was the chief symptom and required morphin for its relief. Late in the disease dyspnea was very annoying. At the time of the last observation the urine was again scant and almost solid with albumin. Stimulating treatment and hot packs availed only for a short time (A to B).

At (C) the patient passed out of observation and died in less than two weeks.

sure through retention of waste, ordinarily eliminated through the kidney. None of the substances, however, which are eliminated by the kidney have a pressor action when injected into animals.

Von Bie¹ cites data to show that there seems to be no uniform relation between the blood-pressure and anatomic conditions of the kidney. He believes the high pressure is the primary disturbance, and that arterial, nephritic and cardiac affections occur only secondarily (see Fig. 55).

PATHOLOGY OF NEPHRITIS

The pathology of chronic nephritis is primarily that of the conditions incident to alterations in the kidney structure, and secondarily (as usually seen by the clinician and the pathologist) that of the many secondary degenerations incident to and largely dependent upon reduced renal function and the changes wrought throughout the body by the high blood-pressure, which is the common accompaniment of the disease and which develops soon after its inception.

Concerning the earlier changes very little of a definite nature is known, and what is believed is more the product of conjecture, partially supported by animal experimentation, than of actual clinical and pathologic study.

The pathology of moderately well-developed and of advanced nephritis, with persistent high blood-pressure, is so protein in its manifestation, involving practically every organ of the body, that it would be impossible as well as superfluous to enter into a discussion of it here.

¹ *Ugeskrift f. Slæger*, Mar. 4, lxxvii, 9.

The post-mortem pathology is of great interest, chiefly because of the light which it throws upon the clinical manifestations, and by serving at once as a check on clinical diagnosis and as a spur to more careful and painstaking study, in the hope that greater clinical ability will eventually decrease the percentage of error which now exists in the clinical diagnosis, as shown in the light of necropsy findings.

The most important single result of post-mortem pathology has been to demonstrate the almost invariable relation of very high blood-pressures to chronic kidney changes. Thus Joseph L. Miller¹ reports that, in Krehl's clinic, of the cases with pressure of 200 or over, 87.4 per cent. showed definite clinical evidence of nephritis, while forty-two of the forty-three cases coming to autopsy showed definite renal involvement.

J. Fisher² reports a review of 550 cases of continuous high pressure. Of the cases above 140, 62 per cent. showed nephritis, while of the cases with pressure over 160, 80 per cent. showed nephritis. Forty-two cases came to autopsy, all of which showed evidences of kidney disease, even those where there had been no clinical evidence of it, though congested kidney or idiopathic dilatation of the heart had been diagnosed.

Roger L. Lee³ reports both clinical and pathologic (autopsy) findings in fifty-three cases seen in the wards of Massachusetts General Hospital, all of whom showed systolic blood-pressure of over 160. He found high pres-

¹ *Jour. A. M. A.*, Oct. 44, 1913, lvi, 14.

² *Deutsch. Arch. f. klin. Med.*, cix, No. 5.

³ *Jour. A. M. A.*, Vol. lvii, No. 15, p. 1179.

sure associated with kidney lesions in thirty-eight cases or 71 per cent. Seven who showed kidney lesions also had a systolic blood-pressure ranging from 165 to 240 mm. Their ages were between twenty and forty-nine years.

High blood-pressure existed with arteriosclerosis in thirty-seven cases or 69 per cent. General arteriosclerosis was associated with lesions of the kidneys in twenty-eight cases or 52 per cent. There was only one case of high pressure with arteriosclerosis without kidney, cerebral, or cardiac lesion; this showed only hypertrophy and dilatation. The patient was sixty years old and the blood-pressure was 210.

Of cases with high blood-pressure without kidney lesion, these were 15 or 28 per cent. Among these seven showed cerebral lesions, four had cerebral hemorrhage and seven showed cardiac lesions. The blood-pressure varied from 175 to 260.

C. Fiessenger,¹ examined 150 patients with high pressure, in 52 per cent. of which he found interstitial nephritis.

Theodore C. Janeway² reports 500 cases of pressure over 170, 150 of whom have died and concerning which the subsequent history and the cause of death were available in 100. Of these, the clinical diagnosis was chronic nephritis in 79 per cent. and arteriosclerosis 4 per cent. He believes that these four cases of arteriosclerosis should be included in the nephritics, as it is unlikely that such cases can persist for long without involving the kidney, and because autopsy statistics show that this is a most frequently overlooked item in clinical diagnosis.

¹ *Bull. de l'Acad. de Med.*, Apr. 16, 1912, lxxvi, No. 16.

² *Jour. A. M. A.*, Dec. 14, 1912, lix, p. 2106.

Causes of Death in High-pressure Cases (Janeway)

	Per cent.
Gradual cardiac insufficiency.....	29
Uremic convulsions or sudden coma.....	15
Chronic uremia.....	20
Cerebral apoplexy.....	14
Acute edema of the lungs.....	4

In this table, which is only partially quoted here, as the chief interest is in the effect of this condition upon the cardiovascular and renal systems, an effort was made to determine the primal cause of the fatal issue, though other causes were not uncommonly associated with it, such as anemia, pneumonia, etc.

SYMPTOMATOLOGY

Signs and Symptoms.—Chronic interstitial nephritis in its well-developed form is usually the result of a gradually progressive process, leading up to a clinical picture which is too well known and too easily recognized to require more than passing comment. It is particularly the early states of this disease with which we are concerned. Our chief effort and desire is to reach an early provisional diagnosis, so that preventive or prophylactic treatment may be instituted at a time when proper management may be reasonably expected to arrest the progress of the degenerative process in the kidneys, and so to indefinitely prolong the individual's period of usefulness and life by protecting the arteries and heart from the increasing strain to which they are invariably subjected, and which in a large percentage of cases contributes largely to the fatal outcome.

Blood-pressure.—A permanent elevation of both systolic and diastolic blood-pressure is the most prominent and

characteristic sign of well-developed chronic nephritis. Sawada¹ states that he has never seen a case of hyper-

BLOOD PRESSURE CHART

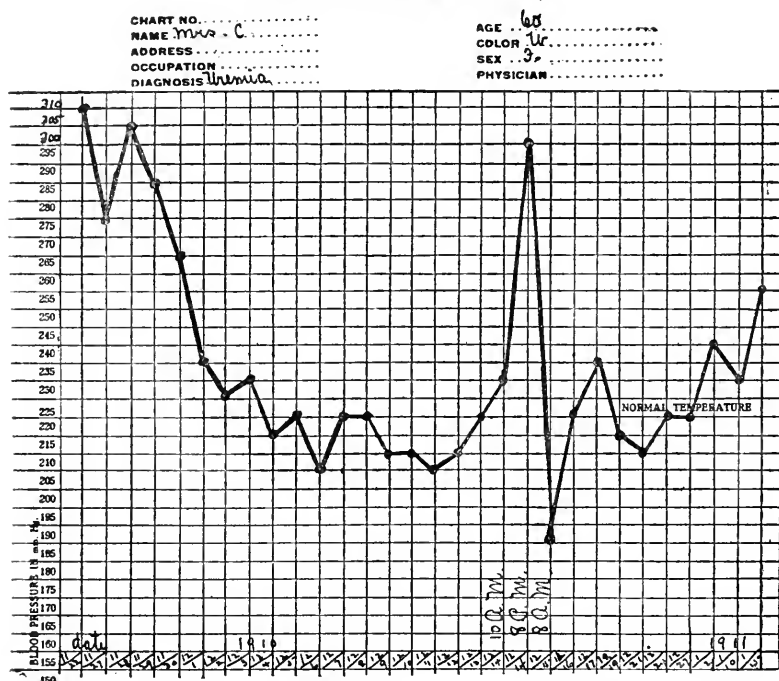


FIG. 56.—Uremic paralysis, resulting from chronic interstitial nephritis. Treatment consisted of hot wet packs, nitroglycerin, 1 per cent. solution, 1 minim every hour, and magnesium sulphate. Treatment stopped on December 1, and sodium nitrite substituted, December 7, nitrite stopped, hot packs continued. Following sudden rise on December 14, thirty-minute pack, 2 minims nitroglycerin, repeated every two hours, resulted in profound fall as shown, with no bad effect. Subsequent treatment consisted of thirty-minute vapor baths and weekly purge, in spite of which there occurred a gradually rising pressure.

tension of more than 170 mm. in simple arteriosclerosis. This limit has been so frequently and conclusively demon-

¹ *Deutsch. med. Wochen.*, 1904, No. 30.

strated to be a safe one that it has been accepted as standard, and is now used by most observers. Romberg¹ considers that persistent high blood-pressure in a suspected case establishes a diagnosis of chronic interstitial nephritis. From the author's experience, in the light of post-mortem findings it seems very doubtful whether a high arterial pressure, from arteriosclerosis or any other cause, can persist over a long period of time without giving rise to the chronic congestion and permanent degenerative changes in the kidneys, with a clinical picture known as chronic Bright's disease. The pressure is higher than that seen usually in any other chronic disease. Sphygmomanometric observations daily confirm this. A systolic blood-pressure of more than 200 mm. (standard cuff) is not uncommon, and I have seen several cases with a reading of over 300 mm. (Fig. 56), two of which have been under observation for several years and will be referred to later. A second salient feature of this disease is that the diastolic blood-pressure does not show a proportionate elevation, but is usually from 60 to 90 mm. lower, thus making an increased pulse pressure (evidence of unnecessary overwork of the heart). Factors such as marked general arteriosclerosis or aortic regurgitation will further accentuate these changes.

In the period of compensation which exists throughout the greater part of the disease it is probable that several factors are separately or jointly contributory to the symptom of heightened blood-pressure. Janeway² has grouped these causes under these heads:

¹ *Kong. f. Int. Med.*, 1904, No. 60, p. 17.

² T. C. Janeway, *Am. Jour. Med. Sci.*, May, 1913, cxlv, No. 5.

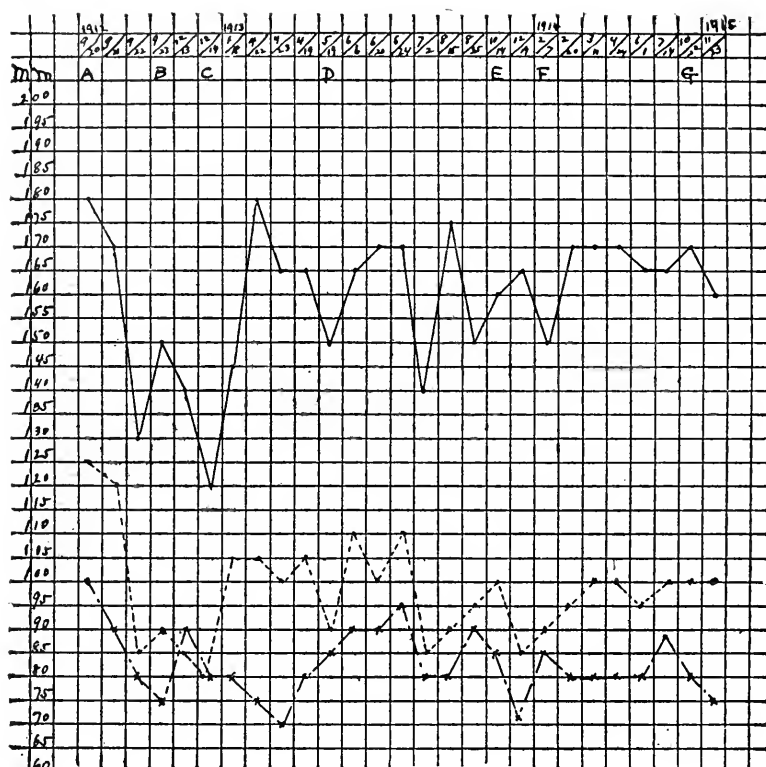


FIG. 57.—Male. Aged sixty-seven. Draughtsman. Chronic interstitial nephritis following chronic alcoholism. Chief symptoms, severe head pain, dimness of vision, dizziness, and ringing in the ears. At first examination there was slight general edema, some pulmonary edema, drowsiness and mental confusion with a typical chronic nephritic urine. The effect on pressure from (A) to (B) was accomplished by eliminative measures including hot packs and purges and was accompanied by complete relief from all symptoms except physical weakness. Dyspnea at (C) was very marked and the pulse was irregular in force and rhythm, although the general condition was fairly satisfactory. Still there was some evidence of muscular weakness and paresthesia in the left arm. (D) Mild return of uremic symptoms, but patient did not lose consciousness and promptly responded to treatment. At (E) patient had eleven roots extracted under gas without untoward effect. At (F) the patient was still in good condition and practically symptom free, although he has given up both work and alcohol and is taking much better general care of himself than heretofore. At (G) there is nothing in the record to show that the patient is handicapped by his kidney condition and is at present living a fairly regular, quiet life.

1. Hypertension may arise through purely quantitative reduction of kidney substance below the factor of safety. This is rare and probably only seen in the later stages of the disease, where myocardial failure contributes largely to this lowered functional capacity.

2. It may arise in connection with some unknown intoxication, which causes disturbances of the central nervous system and which we term uremia. This intoxication is not one of retention, in a strict sense, though it is most commonly present in those cases of advanced nephritis which manifest marked nitrogen retention. Clinically it may be associated with severe acute nephritis.

3. Continued high pressure may arise from primary irritability of the vasoconstricting mechanism from unknown, probably extrarenal causes, which lead eventually to arteriolar sclerosis. In this type the disease in the kidney is the sequence, not the cause of the generalized vascular lesion.

Systolic and Diastolic Pressures.—It is thoroughly understood that any condition which may be included under the head of nephritic hypertension or nephritis presupposes an increased blood-pressure, although this is not invariably the case. The variety of nephritis present determines to a degree the height of the pressure, both systolic and diastolic; thus I. N. Schwartzman¹ analyzing 100 cases of various forms of nephritis found that the systolic and the diastolic pressures were both increased in uremia (see Fig. 57), in acute parenchymatous, in chronic interstitial and in the combined form of nephritis.

¹ *Russky Vrach*, 1915, xiii, No. 28.

We believe that the systolic and diastolic pressure may be increased long before any other signs of chronic nephritis develop, and that high blood-pressure is therefore a reliable early sign of nephritis. A large pulse pressure is generally a sign of good compensation.¹ At the termination of

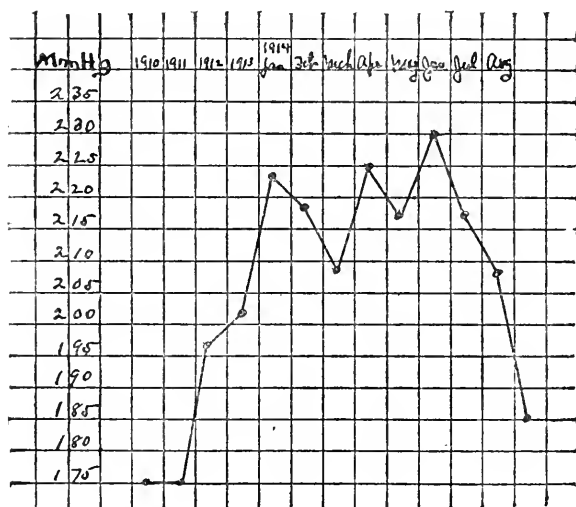


FIG. 58.—Chronic interstitial nephritis. Male. Aged fifty-six. This chart shows the rapidity of the rise in systolic pressure in a case of chronic interstitial nephritis and myocarditis in a patient of fifty-six who failed to appreciate the necessity of care or treatment and was willing to, as he said "take his chances." Cardiac dilatation began when the systolic pressure reached 230 and rapidly advanced. The patient died in an attack of pulmonary edema with systolic pressure 185. Observations before 1914 recorded on chart are yearly averages. 1914 observations show average systolic pressures by months.

a case of chronic nephritis, the systolic pressure tends to fall, due to diminished cardiac activity and the diastolic pressure increases. This is usually a grave sign, pointing to myocardial failure (see Fig. 58). The pulse pressure in nephritis is invariably increased even when the systolic

¹ Warfield, *Am. Jour. Med. Sci.*, cxlviii, 6, 1914.

is not materially above normal. This is evidence of cardiac overactivity plus the constitutional effect of the toxins which have primarily produced or which are secondarily the result of the nephritis. Marked arterial involvement increases this systolic elevation; at the same time increased rigidity holds the diastolic pressure down, so that invariably the pulse pressure is increased. The degree of this increase will depend upon (a) stage of the disease, (b) the degree of cardiac hypertrophy and compensation and (c) the degree and extent of arterial involvement.

Considerable study has been made of the relation of pulse pressure to urinary excretion. It is generally believed that increased pulse pressure increases urinary excretion and Lawrence's¹ studies seem to prove this; on the other hand, there appears to be no relation between the height of systolic and diastolic pressure *per se* and variations in kidney output, although an increase in pulse pressure accompanied by a fall in systolic pressure appears to be followed by diuresis. There also seems to be a relation between the normal systolic, diastolic pulse pressure ratio which normally should be 1-2-3, so that whenever the several pressures approach this normal ratio, irrespective of the height of systolic pressure, there tends to be a rise in urinary excretion.

Urinary Findings in Nephritis.—While text-books, as a general rule, present comparative columns which attempt to show clear lines of differentiation between the urinary findings in various forms of chronic nephritis, it is clinically fully appreciated by close observers that such a differentiation is probably impossible. A single urinalysis is practically

¹ *Amer. Jour. Med. Sci.*, September, 1912.

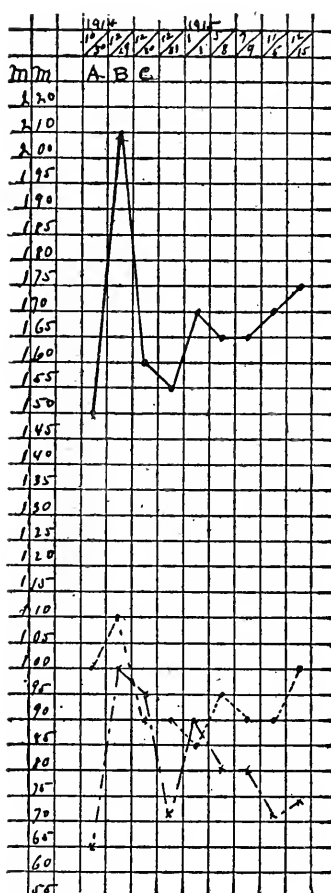


FIG. 59.—Female. Aged fifty-one. Uremic hemiplegia. The first observations (A) were made in the course of a routine examination and no special circulatory abnormality noted. The observation at (B) made several hours following onset of the right hemiplegia which urinalysis and subsequent developments showed to be uremic in origin. Observation at (C) was made immediately after a hot pack. Note the prompt fall in systolic pressure which was affected to a greater degree than was the pulse pressure, which remained far above the normal ratio. In this case recovery was extremely slow and the patient was left with some right-sided disability, although there has been no recurrence of uremia. That the relief afforded is only temporary is suggested by the marked and persistent elevation in pulse pressure together with the tendency of the systolic pressure to rise in spite of continued active treatment.

valueless, whether or not it shows any pathologic variation. Repeated urinalyses are of value when the majority are positive, at least in so far as they indicate alterations in kidney function; but they should not be depended upon where the findings are indefinite. Neither can the urinalyses be depended upon to assist in separating the clinical forms of chronic nephritis thus:

Hewlett¹ reports fifty cases of continued high pressure, usually above 180, eighteen of which showed no urinary changes other than rare traces of albumin or occasional casts (see Fig. 59).

Edema of nephritic distribution is corroborative evidence, but as it is rarely present before heart-muscle failure develops, it cannot be depended upon for an early diagnosis, nor when present does it determine the degree of kidney impairment.

Edema of the lungs may occur as an acute condition during the course of a chronic nephritis. It may, if severe and unrelieved, constitute the terminal process in the case.

This condition is frequently preceded by an abrupt rise in blood-pressure which if detected may ward off the attack by the institution of appropriate measures.

Cardiac asthma may arise under the same circumstances and it is contended by some² that the acute rise in blood-pressure is the cause of the cardiac asthma. Probably the true sequence of events is that the primary disturbance is vasomotor and cardiomotor, the insufficient left ventricle being subordinate, which disturbs the proper working

¹ *Loc. cit.*

² K. Petren and G. Bergmark, *Berl. klin. Wochen.*, Dec. 17, 1910, xlv.

balance between vascular tension, cardiac action and respiration.

Ocular signs are not reliable criteria of the degree of kidney impairment. They may appear early and persist or they may develop late, long after a satisfactory diagnosis has been established.

The *subjective* symptoms differ in no way from those associated with arteriosclerosis and have been fully discussed under that head.

The *cardiac evidence*, often dwelt upon so extensively in dissertations on chronic nephritis, are not truly nephritic at all, as they are dependent wholly upon the arterial condition, and when present are purely myocardial signs and of myocardial significance and should be given this interpretation.

DIAGNOSIS

A diagnostic distinction between the several conditions grouped under the general head of nephritic hypertension and chronic Bright's disease is often impossible. This has already been brought out in the paragraphs devoted to etiology and pathology. The difficulty is further increased by the fact that cases are encountered in all periods of development of the disease, from those bordering on pure hypertension to those in the advanced stages, in which the arterial and the cardiac involvement may completely overshadow the renal.

To be of any value, any effort at a definite diagnosis must endeavor to estimate the degree of nephritic involvement together with the likelihood of a uremia, a cerebral rupture or a heart muscle failure; as it is upon these factors that active and intelligent treatment must be based.

In the diagnosis of any given case it is well to outline the points to be determined, then carefully to weigh them one against the other in the final analysis. Roughly the subject divides itself into a consideration of (a) the general physical condition; (b) the urinary findings; (c) the cardiac adequacy; (d) the arterial involvement; (e) the blood-pressure picture; (f) the renal functional efficiency; and (g) the ocular manifestations.

(a) **The General Physical Condition.**—Much can be determined by a careful physical examination and a thorough history that throws light upon the patient's general efficiency, including mentality, assimilation, nutrition, and general muscular development.

(b) **The Urinary Findings.**—In parenchymatous nephritis the urine picture is very similar to that of acute Bright's disease in which the amount of urine is scant, of high specific gravity, with a relatively large albumin content, a variety of casts; and differing from it only in the usual absence of blood casts, though there may be an occasional red cell, together with hyaline and epithelial casts. Chronic interstitial nephritis and contracted kidney show increased volume of urine with low specific gravity, a scant or absent albumin reaction, while hyaline and granular casts are usually found, if diligently sought, but are often overlooked in routine examination. In the examination of single specimens occasional red cells and epithelia may be found.

(c) **Cardiac Efficiency.**—The functional capacity of the kidneys in cases coming under this head is almost entirely dependent upon cardiac efficiency, as measured by the ability of the heart to maintain a normal circulation against

the elevated blood-pressure which is the result of general arterial involvement including its immediate effect on heart-muscle nutrition. A high pressure with extensive arterial involvement may present a better picture in the presence of an hypertrophied and muscularly competent heart than

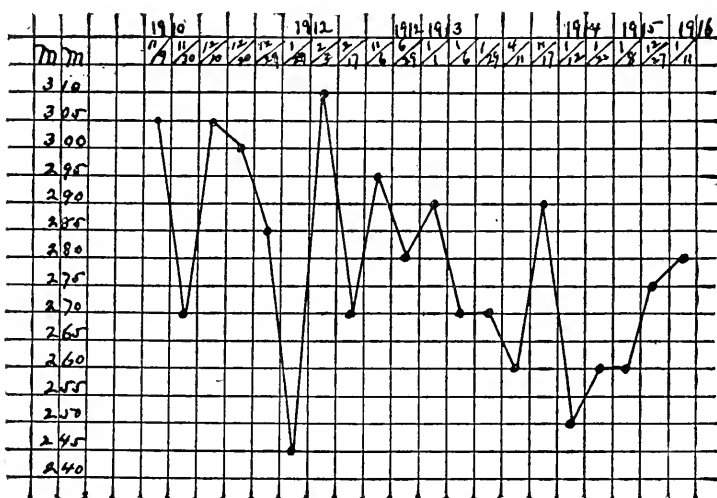


FIG. 60.—Female. Aged fifty-eight. High-grade chronic interstitial nephritis of many years standing. Has had several attacks of chronic uremia with tendency to muscular weakness on left side. The case, which has been under continuous observation for a period of five years, shows the most consistently high systolic pressure ever encountered by the author.

At the last observation, both symptomatically and by physical examination, the patient appears to be in a generally better condition than in 1910. The urine still shows increased volume, reduced specific gravity, occasional granular and hyaline casts and traces of albumin.

a case showing less arterial involvement and a lower pressure accompanied by a poor myocardium. Therefore it is important in every case (see Fig. 60), not only at the first examination but during the subsequent course, to follow closely the muscular efficiency of the heart in relation to the handicap under which it is operating.

(d) **Arterial Involvement.**—We have seen that arterial involvement, as demonstrated by the examination of superficial vessels, is not always a safe criterion upon which to judge the degree of renal change. Arterial involvement may be very irregular in its distribution and we are not justified in condemning the kidneys upon the evidence offered by a study of the superficial branches of the arterial tree. It is also recognized that marked superficial involvement may occur without marked elevation of blood-pressure; at the same time we do not believe that a blood-pressure persistently of 200 or more can exist without some degree of general arterial and renal involvement.

(e) **Blood-pressure.**—It has just been stated that the majority of observers now hold that a systolic pressure of over 170 is gravely suspicious, that a systolic pressure of persistently 200 or more is almost conclusive evidence of kidney involvement (see Fig. 60). This, together with a continuously increased pulse pressure, may be accepted as evidence of renal involvement *per se*, while the higher the systolic pressure and the longer the duration of the high pressure the more likely is this to be true.

(f) **Renal Functional Efficiency.**—However attractive it may appear, from the scientific point of view, to estimate the degree to which the anatomic elements of the kidney are involved, it would seem that the mixed type of nephritis, so frequently encountered clinically, practically disposes of any attempt to arrive at fine diagnostic distinctions concerning the reactions secured by differential function tests. Even in the simplest clinical type of nephritis there is enough general structural alteration to render conclusions as to special functional involvement hazardous.

Therefore as the matter stands, the only methods available for clinical usage are such as furnish information as to the total function of the kidneys, *i.e.*, their excretory capacity.

The original deductions of Roundtree and Geraghty, which have been fully corroborated by subsequent observers, show that the amount of phthalein excreted varies, as a general rule, with the extent of renal damage.

The test is also valuable in cardiorenal cases with failing heart and kidney engorgement. Such a condition is accompanied by a low phthalein output, which rapidly rises as the heart's action improves, so that this change in such a case may be the earliest sign of restoration of compensation, when it becomes of considerable prognostic importance. On the other hand, a persistently low excretory capacity with apparent clinical evidence of cardiac improvement points to a severe nephritis and to a less favorable prognosis.

Efforts to explain the relation of the phthalein test, as seen in practice, to the anatomic conditions revealed in the kidney in necropsy have been made by many, including Thayer and Snowden.¹ Their cases show that in severe chronic nephritis there occurs most uniformly an unusually low phthalein output, which as a rule, unless interrupted by an acute terminal process, decreases steadily up to the onset of uremia, and that excretion may be nearly or wholly suppressed for from only a day or two to a month before death. These authors state that not in all their studies for five years have they met with a good phthalein excretion in a case of chronic nephritis.

There seems to be no question as to the usefulness of this

¹ *Am. Jour. Med. Sci.*, 1914, cxviii, 781.

test, which when periodically employed constitutes a method of value in nephritic and high-pressure states generally. It has been observed that the response to the test is on an average higher in ambulatory than in hospital cases, irrespective of the clinical condition present.

It was first suggested by Miller and Cabot and has been more or less confirmed since, that the excretion of the phthalein grows progressively less with advancing years, irrespective of the kidney condition.

It is a common experience to note that the output is well within the requirements in the majority of cases grouped as nephritis, when accompanied by good cardiac compensation, so that while these observations therefore may not be of great value in diagnosis, they are of undoubted service in the study of nephritis, where they may often furnish the much-needed clue to the presence of doubtful and unsuspected uremic conditions. The phthalein test is also of value in separating the cardiorenal from the cardiovascular cases. Finally from the prognostic standpoint the test offers us great assistance when employed periodically.

The general consensus of opinion now bears out the original conclusions of Folin, Dennis and Seymour regarding this test in relation to the nonprotein nitrogen of the blood, namely, that cumulative phenomena occur only when excretion of phthalein falls below 40 per cent. in the first two hours.

(g) **Eye-ground Examination.**—Ocular symptoms are often among the very earliest signs which are susceptible of detection by careful examination, although in some cases these changes may be greatly delayed. This fact

becomes important, therefore, from a prognostic standpoint because the earlier the diagnosis can be made the more probable is it that good results will be secured from treatment.

Of recent years the relation of retinal changes to blood-pressure and chronic kidney disease has attracted widespread interest. As such examinations, occurring in the course of routine examinations of the eye, have often been the first clue to the presence of chronic renal changes and should therefore indicate the immediate use of the sphygmomanometer, so also should high blood-pressure readings call for an ophthalmoscopic examination. (See Fig. 55.)

PROGNOSIS

Except in the later stages, when all signs point to the approach of death, a prognosis cannot be accurately given until careful observation has extended over a period of time sufficiently long to permit the physician opportunity to carefully weigh the many factors in the case which must, of necessity, contribute to his opinion.

Generally speaking, the earlier the diagnosis the better the prognosis, although there are exceptions to this. The average blood-pressure level is a factor of first importance.

From the standpoint of nephritic involvement as bearing on the ultimate outcome, the prognosis in the case of a robust looking man of fifty or fifty-five, with an average blood-pressure of 200 or over, even with no direct evidence of arteriosclerosis, is not so good as in the case of a man sixty-five or so, with marked evidence of arterial involvement, who is carrying a pressure of between 130 and 160. The former is in daily danger of a sudden total catastrophe,

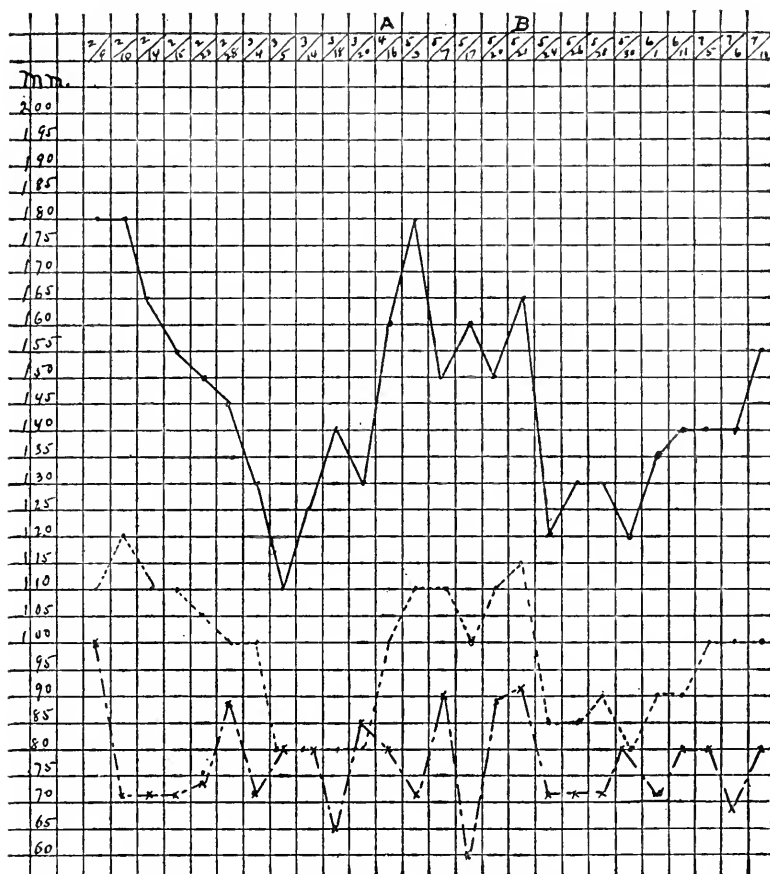


FIG. 61.—Female. Aged fifty-one. Uremic paralysis. First observation was made twenty-four hours after an attack of left hemiplegia of uremic origin. The urine showed albumin and hyaline casts in practically all specimens throughout this period of observation, although less in amount in the later ones. Elimination and sedative treatment produced prompt reduction in systolic pressure to a surprising degree, followed by a less marked fall in pulse pressure. At (A) the patient has so far recovered the use of her lower extremity as to be able to get around with help. The rise at this point was probably due to increased muscular activity and did not give any great concern. At (B) the patient had an attack of tachycardia from no apparent cause, the nurse reporting that prior to my examination the pulse was almost imperceptible, being over 200 and uncountable. Absolute rest plus morphin and ice bags resulted in a prompt fall in systolic pressure. Subsequently the course of the case was uneventful, the slight rise in systolic and pulse pressure being due to gradually increasing physical activity.

while the latter may live on for years with apparent good health.

The most that can be expected from rational treatment is a more or less complete arrest of the progress of the disease, which can be counted upon to delay the fatal termination for a variable period, often for years.

The result of clinical observations and the persistence of certain characteristic changes seen at post-mortem examination indicate that one may, in a certain number of cases, forecast the ultimate termination at least in so far as the forecasting of the variety of death is concerned.

Janeway¹ has reported his findings in a long series of cases, from which the following has been largely taken.

In Janeway's series twelve out of twenty-nine cases which died of cardiac insufficiency, began with dyspnea on exertion. On the other hand, out of thirty-two patients, who noted polyuria or nocturnal urination at the beginning of their illness, seventeen died of chronic and seven of acute uremia while only eight died of cardiac insufficiency.

Duration of Illness in Relation to Causes of Death (*Janeway*)

Causes of death	Cases	Duration				
		Average		Longest, yrs.	Shortest	
		Yrs.	Mos.		Yrs.	Mos.
Cardiac insufficiency....	26	3	10	10	4
Acute uremia.....	14	3	8	5
Chronic uremia.....	21	3	1	7	4
Cerebral apoplexy.....	14	4	6	11	11
Angina pectoris.....	3	4	3	6	3	6
Acute edema of lungs....	4	3	4	1	8

¹ *Loc. cit.*

The relation of headache and subsequent uremia is most striking. Of fifteen patients complaining of headache, eight died of chronic uremia (Fig. 61), two of apoplexy, and only one of heart failure.

Early ocular symptoms were most frequent in those who died of chronic uremia, in five out of eight. Early hemoplegic attacks occurred thirteen times in four patients who finally died of apoplexy.

SUMMARY

1. Early dyspnea of either type in a patient with high pressure indicates marked danger of cardiac insufficiency. In such patients the disease should be treated as a cardiac disease.

2. Anginoid pain, even when of marked severity, occurring in persons with high blood-pressure, does not make the prognosis worse than other cardiac symptoms. The majority of these patients will not die of angina.

The Relation of Prominent Early Symptoms with High Blood-pressure to Causes of Death (*Janeway*)

Symptoms											Causes of death	No. of deaths
Dyspnea on exertion	Paroxysmal dyspnea	Edema of lungs	Anginoid pain	Edema of legs	Polyuria	Headache	Visual disturbances	Hemiplegic attacks	Loss of flesh	Neurasthenic symptoms		
24	13	1	5	7	8	1	1	2	2	1	Gradual cardiac insufficiency	29
7	2	0	1	0	7	2	1	2	3	3	Uremic convulsions or sudden coma	15
5	1	2	3	0	11	8	5	4	5	1	Gradual uremia	20
6	2	0	2	2	2	2	1	4	0	4	Cerebral apoplexy or its results	14
1	1	0	2	0	0	0	0	0	0	0	Angina pectoris	3
2	2	1	1	1	1	0	0	0	1	1	Edema of the lungs	4
3	3	0	1	1	3	2	0	1	3	6	Other causes	15
48	24	4	15	11	32	15	8	13	14	16		100

3. Polyuria, nocturnal frequency, marked headache, or visual disturbances in a patient with high blood-pressure, especially if the patient is below fifty years, should make the prognosis very guarded, for uremia is a frequent mode of termination of these cases.

TREATMENT

From the first inception until the very end, the treatment of chronic high pressure and chronic nephritis should be of the individual and not of the disease. This important fact cannot be too frequently or too positively repeated because there is still a large number of practitioners who are endeavoring to treat solely the kidney condition or one or more of the many symptoms or secondary complications with which all are familiar. One should never forget the fact that nothing can be done to remove the existing degenerations, while much can and should be done toward relieving undue strain where such is found to exist, by promoting elimination, favoring cardiac action and in general, regulating the patient's mode of life, in an endeavor to readjust it to existing conditions. Any other method may not only be useless but may actually work harm.

It is absurd to treat any case directly for the blood-pressure elevation, by measures directed toward actively reducing the high pressure, as it has been shown by many, that in point of longevity¹ but little difference is found between those whose systolic pressure rises above 200 and those that range between this and 150.

Everyone should be fully prepared to accept the statement that very high blood-pressure may exist as a direct

¹ T. C. Janeway, *Arch. Int. Med.*, xii, No. 6, p. 755.

response to physiologic stimuli, and that such a reaction may be entirely natural, if not essential. Here it becomes physiologic.

It may be said, I think without question, that the most important item in the treatment of chronic nephritis is the preservation of cardiac compensation. Neither is there any question but that the elevation in systolic pressure while bearing a direct casual relation to the cardiac hypertrophy, also operates in conjunction with it in forming a compensatory mechanism which alone is able to maintain an adequate kidney function. They are, consequently, essential to the preservation of life, and should be protected by every hygienic and dietetic safeguard.

In connection with treatment by hygienic and dietetic measures, it is often most strikingly demonstrated that such measures, if they result in relief of distressing symptoms, cause a reduction in pulse pressure and that this occurs even when the systolic level is not materially affected. In my own work I have come to look upon this alteration as auguring good, even when, as already mentioned, the systolic level remains the same (see Fig. 62).

I cannot agree with those authorities who divide the treatment of this condition into (a) correction of the tension and (b) the administration of specific drugs. It is rare except in emergency that such drugs are required (as to relieve threatened death from pulmonary edema, acute dilatation of the heart or apoplexy) and they may and frequently do cause harm. Therefore, nitrites should be reserved for emergency and then given only when carefully checked by the clinician. Even then they are often of little value because of the uncertainty of their action.

The appearance of dropsy in chronic nephritis with high pressure almost invariably presages cardiac failure. At this stage the digitalis bodies become our sheet anchor of treatment. They should not be withheld because the blood-pressure is high, as they have been found to act just as well, or even better, in the presence of high pressure than with a decreased pressure.¹

That the ingestion of sodium chloride favors fluid retention is well known, so that its reduction should be encouraged, in any case where the presence of edema fails to respond to other methods, although I am not yet willing to state that salt restriction should be included among the dietetic measures in every case.

Space will not permit of an extended discussion of the many dietetic, hygienic and therapeutic measures which may be employed in the management of the disease. Those desiring specific information relative to the variety and to the effect of such measures will find them briefly discussed in Chapters XXIII and XXIV.

¹ A. R. Elliott, *Jour. A. M. A.*, Nov. 21, 1914, lxiii, 21, 1878.

CHAPTER XIX

CARDIAC DISEASE: MYOCARDIAL, VALVULAR, AND FUNCTIONAL. MYOCARDIAL INSUFFICIENCY

Significance of Term.—From a practical standpoint it would seem advisable to employ the general term myocardial insufficiency, to the exclusion of all others, when discussing from a clinical standpoint the pathologic changes which may occur in the heart muscle; for, while we recognize pathologically a sharp line of demarcation between acute and chronic inflammation, and between fatty degeneration, fibroid degeneration or fibrosis, senile heart and chronic cardiac insufficiency, in the majority of cases there is no way by which these various conditions can be distinguished from each other clinically. Any attempt to separate the various forms of myocardial change, by a clinical study of the case, is merely an exhibition of ignorance, for the symptoms supposed to indicate different forms of myocardial disease may be caused by the same pathologic conditions. Also various pathologic changes may give rise to identical trains of symptoms, so that all efforts to clinically classify them must necessarily fail.

Chronic myocarditis causes weakness in the cardiac power, irrespective of the cause of the pathologic change in the heart muscle. This is evidenced clinically by an inadequacy of the circulation either during rest or following a demand for increased power.

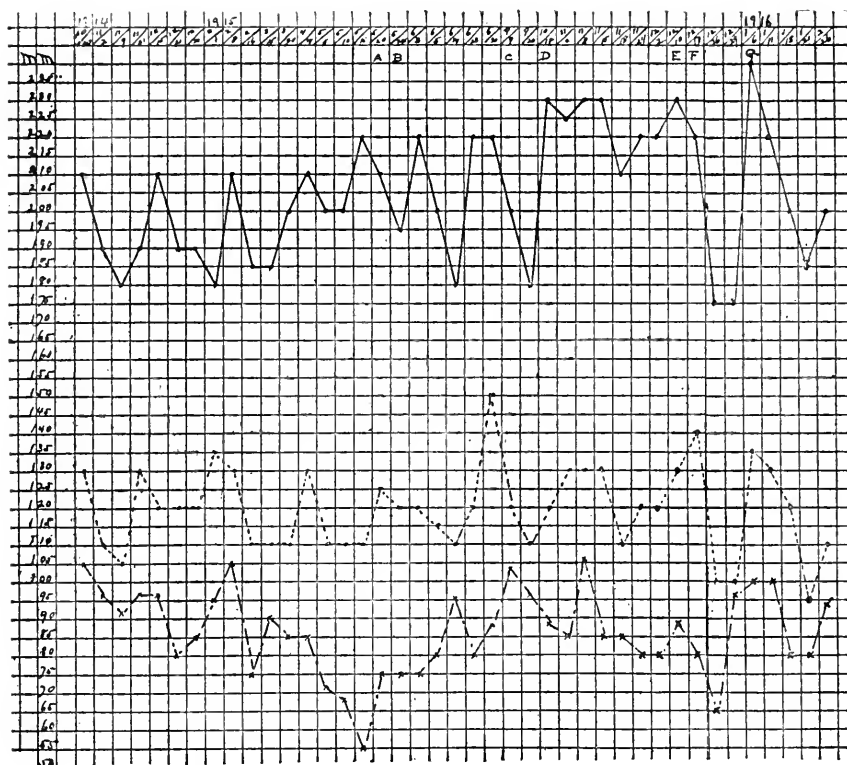


FIG. 62.—Male. Aged sixty. Retired stock broker. Cardiovascular-renal disease. Myocardium in fair condition, arteries moderately rigid. The height of systolic pressure and large pulse pressure are characteristic of arterial rigidity and cardiac overaction. The remissions are unexplained except on the grounds of varying excitement and activity. Measures directed toward relief of high pressure very unsuccessful, although relief from dyspnea, insomnia and early morning headache was quite marked, and has continued in spite of the gradual average rise in systolic and pulse pressures. (A) Large doses of tincture of aconite failed to reduce pressure. (B) An attack of acute rhinitis occurred. (C) General condition good. Has spent summer at seashore resort. (D to E) Not under any treatment. Absence of all unpleasant symptoms and general condition excellent. Note the stationary pulse pressure in spite of moderate variations in both systolic and diastolic. (F) Attack of grippe. Confined to bed until 1-16-16. Attack rather severe with marked toxemia and prostration. Note effect of treatment and rest upon systolic, diastolic and pulse pressures. (G) Note sharp rise in systolic as result of extraordinary exertion following severe infection, accompanied by dizziness, insomnia and cough. Subsequent treatment directed toward circulation including the use of strychnin and caffein by mouth.

Etiology.—Chronic myocardial insufficiency is a condition of the heart muscle resulting usually from some disturbance in nutrition of the heart muscle. Leslie Thorne¹ says that two of the most common forms of muscle degeneration resulting from hypertension are atheroma and fatty degeneration.

The *modus operandi* of degeneration is probably that of a disturbed blood-supply to the heart itself, due to a narrowing of the coronary arteries. It is therefore essentially a chronic progressive process and, from the intrinsic nature of the change, when once the process has become fairly well started it is but slightly amenable to treatment.

Bruce² dwells upon the frequency of cardiac degeneration, associated with glycosuria, and also the frequent relation of gout to chronic myocarditis.

A most important factor, never to be forgotten in this strenuous age, is the effect of the constant strain of responsibility upon business men, legislators, professional men, etc., where we find the development of high pressure particularly common, resulting in cardiac enlargement, with more or less insufficiency, all of which denote the beginning of the end, unless the overstress be reduced.

Belonging to the same class of cases are those due to indiscretions in diet and sedentary habits with insufficient exercise. Here the intra-abdominal vessels are subjected to abnormal and prolonged strain, which leads in time to sclerosis of their coats, to increased blood-pressure, cardiac overwork and eventually to degeneration of the myocardium (see Fig. 62).

¹ *Lancet*, June 4, 1910.

² *Lancet*, July 15, 1911.

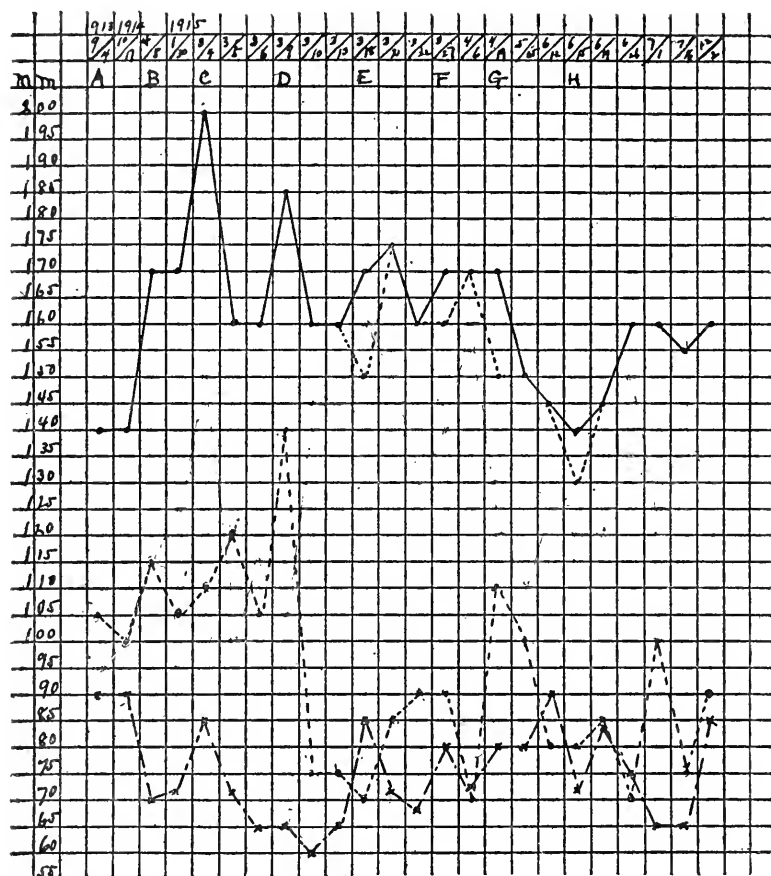


FIG. 63.—Female. Aged seventy-three. Chronic uremia and myocarditis. Observations from (A) to (B) were made in the course of routine examination, following a slight injury. It was noted that the patient's mental condition was becoming clouded and her reflexes were exaggerated, she complained of being smothered and dizzy with persistent ringing in the ears. The observation at (C) was made during a mild attack of uremic coma without convulsions, which was relieved by the usual eliminative treatment. At (D) the kidney excretion was reduced to under 15 ounces in twenty-four hours and was highly albuminous. This state gradually responded to digitalis medication. At (F) the uremic condition had largely disappeared and was replaced by dilated and incompetent heart. The blood-pressure and an examination of the circulation at this time showed a rather characteristic evidence of heart muscle weakness, in that it was easy to distinguish

Fatty degeneration of the heart is due in most cases to the same conditions which cause atheroma of the aorta and disturbance in the coronary circulation. It is also one of the natural results of advancing age, where it is generally dependent upon the long-continued action of such irritations as chronic auto-intoxications, habitual use of alcohol, toxic effects of tobacco, coffee, etc.

Chronic disease of the kidneys, by increasing the resistance in the arterioles raises blood-pressure; this in turn produces general arteriosclerosis, from which the coronary arteries are not exempt, lead inevitably to myocardial degeneration (see Fig. 63). Less commonly we find fatty degeneration following protracted wasting diseases, exhausting discharges or anemia from repeated losses of blood. Acute fatty degeneration usually results from the toxins of diphtheria and other acute infectious processes, and occasionally from phosphorous or mercurial poisoning.

From the clinical standpoint one need not consider any other phase of the subject, as the insufficiency is largely a mechanical condition and its effects are shown chiefly in disturbances of a dynamic nature, presenting diminished cardiac energy on the one hand and increased demand for energy on the other. In considering this condition we

two apparent systolic readings, *i.e.*, the first tone while distinctly audible, almost immediately disappeared, and was then replaced by a false first tone as shown at the apex of the dotted line (*E*). This was actually the beginning of the third phase and was caused by an absence of the second phase. The same condition occurred at (*F*), (*G*) and (*H*) and these changes were always coincident with extreme weakness, cardiac arrhythmia, orthopnea and pulmonary edema, and at the same time the kidney excretion in twenty-four hours was variously recorded at from 10 to 16 ounces. At the last examination the patient showed more the effect of myocardial than a renal involvement, as shown by weakness in the second and third auscultatory sounds and the tendency to a slight average reduction in systolic pressure together with a slight rise in the average pulse pressure.

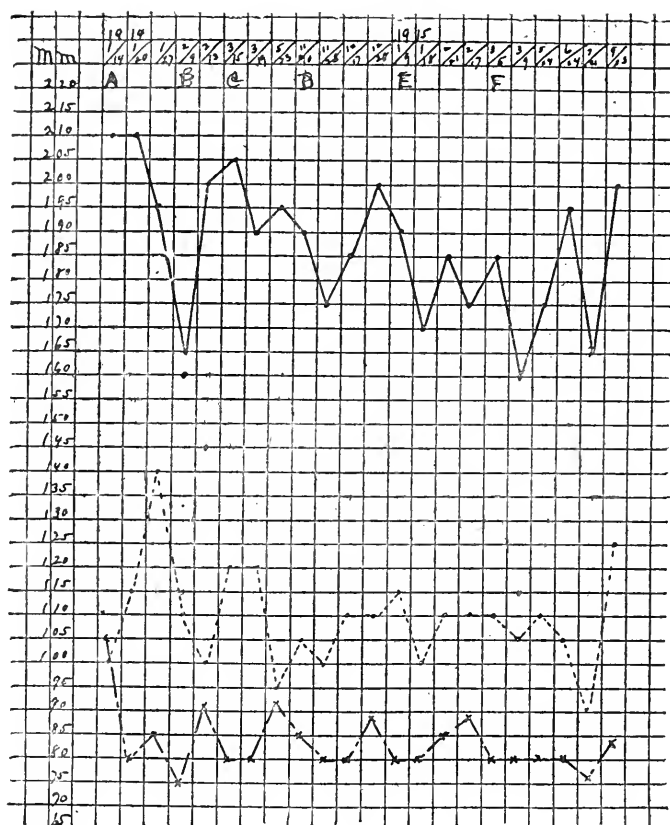


FIG. 64.—Female. Aged fifty-five. Apparent age sixty-five. Chronic myocarditis and autotoxemia. Has long been a sufferer from the effects of extensive pelvic inflammation and has had several operations. Consultation on account of symptoms suggestive of gout, developed evidence of cyanosis, dyspnea and cough. Examination of the urine showed low specific gravity, traces of albumin, granular and hyaline casts and red blood cells. The blood-pressure was 210 mm. Hg. The arteries were moderately fibrous, the heart muscle sounds were poor and there is a faint systolic murmur over the body of the heart. Hygienic measures and warm baths were instituted. The effect of these measures on blood-pressure (A to B) seemed to justify the assumption of a large autotoxemia element. Return to ordinary habits, which have always been active, probably accounted for the rise at (C) although the patient was at this time symptom-free. At (D) the patient complained of dyspnea on exertion, a feeling of fullness in the chest, and tingling and weakness in left arm, which were

cannot confine ourselves strictly to a study of these hearts which are doing less than the average amount of work, as such hearts may actually be over-exerting themselves, but handicapped by leaky valves or high arterial pressure, they are *forced* into this class. We must make a distinction between inefficiency and insufficiency. Hearts may be insufficient because they have a lessened reserve and inefficient because they have an excessive amount of work to perform.

Definition.—**Myocardial insufficiency** (Fig. 64) may be described as the condition when a heart with normal valves does not possess sufficient power to drive the required amount of blood with the normal velocity through a normal arterial system.¹ This is a positive intrinsic vital defect. It also exists when valvular disease imposes a burden of leakage greater than the heart is able to compensate and when high arterial pressure demands additional energy to maintain circulatory equilibrium. Under these conditions the insufficiency is relative, mechanical or extrinsic.

Such a clear-cut, positive distinction can seldom be made in practice, as sooner or later all cases will be found to participate in both.

Myocardial Sufficiency.—According to Gifford² the normal heart of a normal individual under thirty years of

relieved by rest and hot packs. At (*E*) the symptoms returned and dyspnea became more marked. At (*F*) general condition good in the absence of special measures. There is nothing further important in subsequent history. Beneficial effect of treatment shown by slightly lowered general average pulse pressure which should, under proper management, be maintained for an indefinite period.

¹ U. G. Gifford, *Penna. Med. Jour.*, September, 1915, xviii, 12, p. 913.

² *Loc. cit.*

age with the body at rest, does not expend more than one-twelfth of its potential or reserve energy. At moderate work one-fourth to one-third is used, while the full capacity is demanded only in the most severe forms of exercise demanding great endurance. With the body at rest 60 c.c. of blood is expelled at each systole, at the rate of seventy-two times per minute. Additional demands, the result of exercise, can, according to Plesch, drive the heart to deliver six times this volume in the same interval of time. This is the factor of safety, encroachment upon this and reduction in it being the most characteristic features of myocardial insufficiency, and in proportion to this encroachment is the myocardium found insufficient.

Varieties of Insufficiency.—In the *intrinsic* vital form of myocardial insufficiency the cardiac muscle has less than normal power to respond to the increased demands, while in the *extrinsic* mechanical form (valvular disease and high arterial pressure) the heart is handicapped by the load it carries, and in both classes the factor of safety is reduced in proportion to the severity of the existing conditions.

Secondary Factors Contributing to Incompetency.—The heart under the above conditions cannot at any time or under any circumstances respond to the demand for additional effort, neither can it get the normal amount of relief that bodily rest should afford. The inevitable and unfailing result of these changes is impairment of myocardial nutrition, while in addition it also suffers from insufficient coronary circulation, which is the result of an improper oxygen content of the blood supplied to the heart and abnormal function of this blood, due to excess of products of waste and fatigue toxins.

Effect of Strain upon Insufficient Hearts.—While a strong heart tends to decrease in size during exercise, the weakened heart tends to increase, or in other words to dilate. This is due to a deficiency in tonus; consequently a heart in which the muscle is diseased will dilate upon comparatively slight exertion. In cardiac degeneration there is always diminished tonicity, therefore diminished tonus is an important factor in the production of permanent dilatation.

In this connection the venous pressure plays an important part, as venous pressure is increased by exercise, and is particularly high during straining, heavy lifting, etc., this is frequently a factor in the production of overstrain, because a high venous pressure keeps the right ventricle dilated, and if the tonicity is low, the heart muscle will remain dilated. According to the researches of Louis M. Warfield¹ the most important factor in the production of chronic dilatation is constant repetition of the strain. Even a mildly diseased heart may recover from considerable strain, provided the strain ceases quickly or if time is allowed for the heart to return to normal size before the second strain occurs. On the other hand, if repeated strain occurs to a heart already dilated, having low tonicity, then permanent damage results. The border line between true heart failure and complete recovery depends to a large degree upon the period of rest after strain.

Heart Failure.—Broken compensation does not occur in a normal heart, however severe the exercise may be. A normal heart muscle is, however, capable of becoming

¹ *Interstate Med. Jour.*, p. 994, 1911.

acutely dilated. In this condition the pulse may be so rapid that it cannot be counted, and nausea and vomiting may occur. In sudden heart failure the blood-pressure falls rapidly; I have seen the systolic pressure drop 30 to 40 mm., and the diastolic 20 to 25 mm., in less than three minutes. The patient may faint from the exertion, but so far as we know, such hearts return after a time to normal size and by virtue of the normal tonicity of the heart muscle no permanent damage results. Even hearts with evident valvular lesions do not break down, except temporarily, unless the ventricle is diseased. The usual symptoms of heart failure occur often without warning, when the patient is seized with sharp precordial pain accompanied by faintness and dizziness, following which he may sink back in his chair or fall to the floor and expire before any assistance can be rendered.

Symptomatology of Chronic Myocardial Insufficiency.—

One of the earliest manifestations of myocardial change is the development of peculiarities in rhythm. Careful investigation will often show that the function of rhythmicity has been interfered with, causing intermittence, irregularity and extra contraction, or extra-systoles. This means damage to the heart muscle.

F. R. Nuller¹ is of the opinion that since we so frequently find arteriosclerosis in failing hearts in later years, in those who earlier showed extra-systoles, we should pay more attention to this symptom, as it probably represents a very early stage in the evolution of this disease.

In many cases beginning myocardial change may be perceived by studying the two aortic tones, as the first sound

¹ Harvey Lecture, 1906-07.

in the aortic area may be unchanged, weakened or accompanied by a murmur, while the second sound may be either intensified or diminished. A systolic aortic murmur occasionally may be due to a blood state, but in a person of middle age it is strong evidence in favor of alterations in the aortic wall and in the myocardium. Should these findings be associated with thickened peripheral arteries and an elevated blood-pressure, slight but persistent, the conclusion that myocarditis is present is warranted, especially when in addition there have been subjective signs of muscular weakness. Where the transverse diameter of the heart can be shown to be increased, the diagnosis of a clinically weakened and insufficient heart is assured.

Not infrequently the degenerative process progresses without symptoms and is not discovered until an attack of dyspnea or fainting occurs or a paroxysm of angina pectoris proves immediately fatal.

As a result of feeble circulation or of venous congestion, or the development of emboli, many other symptoms and signs may appear in individual cases which, if the physician is on his guard, may readily be traced to their true source.

Diagnosis of Myocardial Efficiency.—From the foregoing it is evident that the determination of the degree of muscular efficiency of the heart, either simple or complicated with valvular defects and high arterial pressure, is far from a simple problem, but one which must be attacked carefully, requiring more than average diagnostic skill and ingenuity.

Many investigators now as in the past are vigorously attacking this problem, studying the effects of postural

changes and physical exercise on the systolic, diastolic and pulse pressures, on the pulse rate, and on venous pressure.

A brief review of the normal effects of exercise on the heart and circulation will serve to bring out the important diagnostic features of the so-called functional tests.

Effect of Muscular Work on the Heart.—Regarding the effect of muscular exertion upon blood-pressure, we believe that in normal hearts there is a rise in systolic pressure and a less proportionate rise in diastolic pressure with the accompanying increase in pulse pressure; and that the more habituated the individual is to performing the work (as in training) the less the tendency for a marked systolic rise to occur, as we find that in highly trained athletes the systolic pressure may fall, though the pulse pressure will have increased. In the average individual the primary systolic rise is followed by a fall to or below normal upon the cessation of effort. The more strenuous the work, the sharper the rise, but following spasmodic as opposed to regular efforts, the speedier and greater will be the fall. This depression in pressure after exercise has been variously ascribed to fatigue, to vasomotor insufficiency and to temporary cardiac dilatation.

In well-compensated valvular lesions and in neurotic hearts the effect of exercise is the same as in normal individuals, and the average rise according to Buttermann is 13 mm., whereas in myocardial cases, instead of the rise there occurs a fall varying from 3 to 13 mm.

Effect of Postural Change, without Muscular Exertion upon Arterial and Venous Pressures.—Barach and Marks¹ summarize their findings in normal circulations as follows:

¹ J. H. Barach and W. L. Marks, *Arch. Int. Med.*, May, 1913, p. 485.

1. When the element of muscular effort has been eliminated, change of bodily posture from the erect to the horizontal will cause an increase in the systolic pressure, a decrease in the diastolic pressure and an increase in the pulse pressure.

2. After five minutes in the horizontal posture, when the subject is returned to the erect posture, the systolic pressure will diminish, the diastolic pressure will increase and the pulse pressure will diminish. It will be noted that in both instances the pulse pressure follows the same trend as the systolic pressure.

3. Change of posture from the erect to horizontal caused a fall in the venous pressure.

4. Change of posture from the horizontal to erect caused an increase of the venous pressure.

It will be noted that the venous pressure follows the same trend as the diastolic pressure.

Change from erect to horizontal causes in the majority of the cases a decrease of the systolic pressure and an increase of diastolic pressure.

General Summary

	Erect	Horizontal	Erect
Maximum.....	×	+	—
Minimum.....	×	—	+
Pulse pressure.....	×	+	—
Venous pressure.....	×	—	+

In Heart Cases.—Change from the erect to the horizontal without effort caused the systolic pressure to increase in five cases. From the horizontal back to the erect a fall occurred in six and a rise in two. The diastolic, in change from the erect to the horizontal, fell in eight and

rose in one while in the reverse the diastolic pressure rose in seven and fell in one, while the rise was to a higher level in five out of eight.

Relation of Pulse Pressure to Venous Pressure.—The tendency in the normal is for a simultaneous increase in

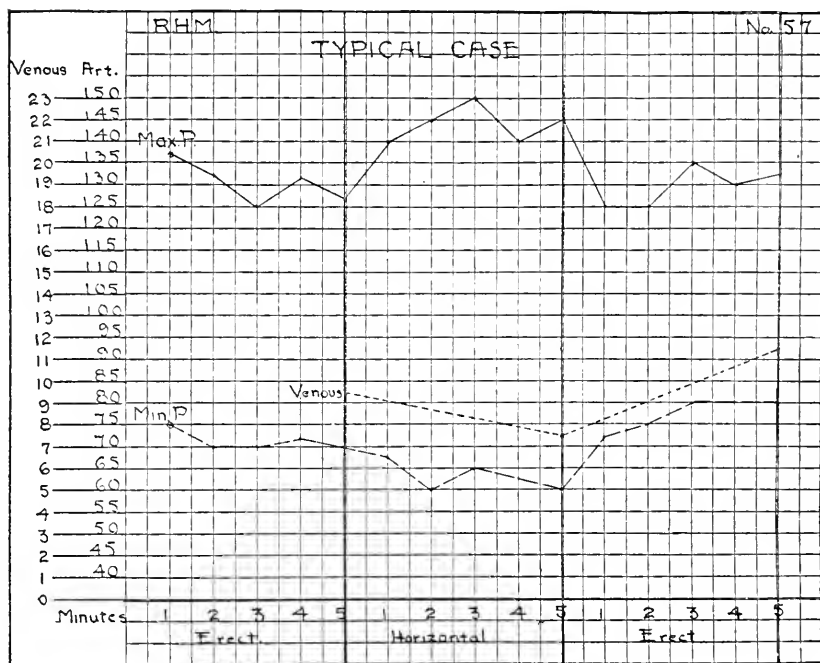


FIG. 65.—Chart showing maximum and minimum pulse pressure in the erect and horizontal position in a typical case (*Barach and Marks*).

both. In heart cases, irrespective of the lesion, the response is similar.

It is well known that a fall in pressure of 10 mm. or more in change of posture from the recumbent to the erect is a sign of poor vasomotor equilibrium. But this does not indicate whether the defect is functional or organic.

Katzenstein's Test.—This test has been employed to determine the degree of functional capacity of the heart and is based upon the reaction of this organ to compression of both iliac arteries, as it has been found that in the higher animals, including man (possessing normal hearts), such pressure causes a rise in systolic pressure of from 5 to 15 mm., with a stationary or diminishing pulse rate, if the obstruction is maintained for several minutes; while in diseased but hypertrophied hearts, with good compensation, the blood-pressure rises to 40 or more millimeters while the pulse rate is unchanged.

Graupner's Test.—This is based upon the physiologic fact that a given amount of exercise, such as ten bending movements, or running up a flight of stairs, causes both an acceleration in the pulse rate and a rise in blood-pressure, but the latter does not occur coincidentally with the former; or if, as in some cases, the pressure does rise first, it fails to rise again after the pulse has returned to normal. It is this secondary rise which indicates a good heart muscle. A not too seriously affected heart may show a rise in blood-pressure immediately after the exertion, but with the slowing of the pulse, the pressure will be found to have fallen to a level lower than before the experiment. The sphygmomanometer is required for an accurate demonstration of these changes in pressure, which may be recorded in definite units of measure for future reference and comparison.

Shapiro's Test.—This is based upon the alteration in pulse rate occurring in normal individuals by change of posture from the standing to the recumbent. Normally, the number of pulse beats per minute is from seven to ten less in the recumbent position, but when chronic myo-

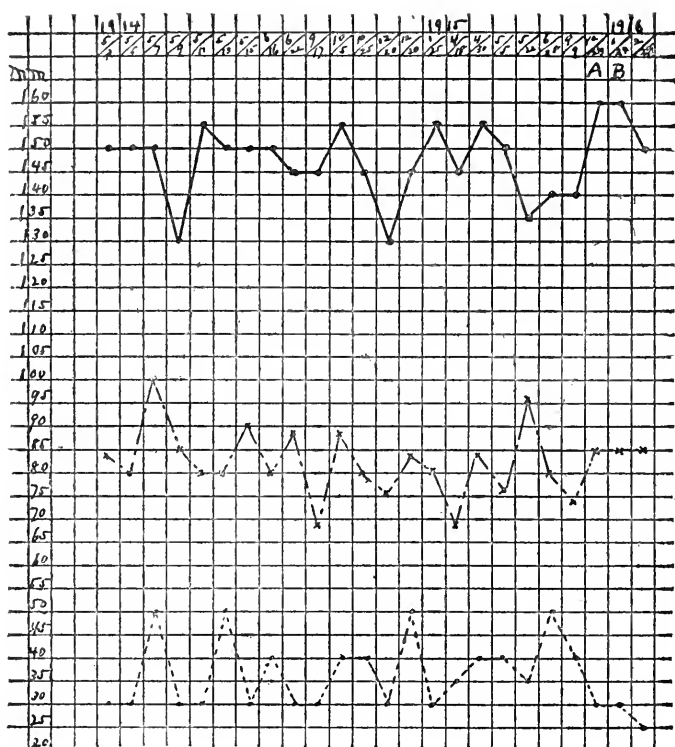


FIG. 66.—Male. Aged twenty-three. Musician. Typical aortic regurgitation. Transverse diameter of heart over 8 in. Characteristic signs of this disease present. Observations have all been made during period of compensation and show usual height of systolic pressure. Characteristic extremely low diastolic and large pulse pressure. (A) Observation followed a period of dyspnea with palpitation and subjective irregularity, the result of mental excitement due to illness in family. (B) Has had another attack of rheumatoid pains throughout body, is short of breath, has a poor appetite and is constipated. Heart is irregular. Note the change in pulse pressure at this point, which may in part be accounted for by slight overexertion and mental worry, although the difficulty in determining the diastolic point by auscultation in this class of cases may have contributed.

carditis develops this difference tends to disappear, so that in seriously weakened hearts the pulse may be as rapid in the recumbent as in the standing posture.

Cautions.—It is not advisable to apply Katzenstein's or Graupner's tests to patients with excessively high blood-pressure, in those of apoplectic tendency or in those with high-grade arteriosclerosis. The tests are unsafe in those with a systolic pressure of 200 mm. or over. In such cases there is danger of ocular or cerebral hemorrhage or acute dilatation of heart.

CHRONIC VALVULAR AND FUNCTIONAL CARDIAC DISEASES

General Considerations.—From the clinical standpoint the chief interest in all chronic valvular lesions of the heart, irrespective of the etiology, centers around the condition of the heart muscle, *i.e.*, functional efficiency. A functionally efficient heart is sufficient to the needs of the circulation, so that any treatment directed toward the heart will be in an effort to promote and maintain muscular efficiency or to guard the insufficient heart muscle from overstrain. While we may be interested as clinicians in the effect of a particular valvular lesion or disturbance in the neuromuscular mechanism of the heart, we are as therapists most particularly interested in the myocardium. In every case the most important question to be answered is: What is the degree of muscular efficiency? Insufficiency is not wholly incident to a valvular imperfection, but is dependent to a great degree upon cardiac muscular efficiency; that is, the yet remaining portion of potential or reserve force that may in time of need be called out.

It has already been noted that a normal circulation presupposes a normally acting heart, while on the other hand, abnormality in the heart's action is promptly manifest in the circulation, so that in any consideration involving the condition of the heart we must include a thorough and comprehensive study of the circulation, directing our attention primarily to the state of blood-pressure and the character of the pulse. It is self-evident that to consider this subject intelligently one must have a knowledge of the normal blood-pressure and its variations. We must appreciate the significance of a mechanical hypotension as referred to in detail on page 238 and the various forms of hypotension discussed on page 236.

Blood-pressure in Heart Disease.—Owing to the almost unlimited power of the heart to accommodate itself to a great variety of external and internal influences, both of a temporary and of a permanent nature, such as occur in valvular and myocardial diseases, it is surprising that blood-pressure observations often show but small variations, and it is not uncommon to meet advanced cases of myocardial and valvular disease in which the alteration in the systolic blood-pressure is insignificant. The conditions usually responsible for elevated pressure in chronic valvular disease are arteriosclerosis and chronic renal disease. The one exception to this is aortic regurgitation. Increased blood-pressure is essentially a vascular and myocardial and not a valvular phenomenon.¹ It will be seen, therefore, that in valvular lesions the actual blood-pressure readings are a reflection of existing secondary conditions, of a progressive nature, rather than of the cardiac lesion

¹ V. T. Korke, *Lancet*, Dec. 21, 1911, clxxxi, No. 4605.

itself. Korke¹ reports a large series of observations made (under carefully controlled surroundings) of the respiration, pulse rate, sphygmographic tracings, besides complete blood-pressure readings, from which he concludes that in chronic valvular lesions of the heart due to the usual causes, the blood-pressure is normal or slightly above, whereas in aortic incompetence, complicated with anginal attacks, the blood-pressure, contrary to the usual belief, is often subnormal during the periods between the attacks of angina.

In valvular lesions complicated with chronic nephritis and arteriosclerosis the significance of the high pressure is explained by the accompanying phenomena.

AORTIC INSUFFICIENCY

In well-compensated aortic insufficiency the systolic blood-pressure is usually found to be somewhat elevated (see Fig. 66) ranging between 130 and 160 mm. (Korke's observations do not agree with this.) Following the onset of this lesion the circulatory efficiency of the heart is probably maintained by reflex vascular stimulation and later by changes in the myocardium, as shown by a great left ventricular hypertrophy, so that as long as compensation is maintained the systolic pressure remains above the average level. With the advent of cardiac muscle failure it tends to fall, rising again if the therapeutic measures employed prove effectual.

In discussing the persistent arterial heart sound in aortic regurgitation, which until recently has been a stumbling block in the determination of the diastolic

¹*Loc. cit.*

pressure, Taussig and Cook¹ state that, in spite of the prevailing opinion, the persistent arterial sound is not pathognomonic of aortic regurgitation, often being absent in this disease and occasionally present in others.

Prognosis.—In heart affections, particularly valvular, a rapid and persistent reduction in systolic pressure is always an unfavorable sign, showing that the heart is beginning to fail. The same may be said of a diminishing pulse pressure, while a slightly elevated systolic pressure may be looked upon with favor.

Special Methods of Determining Aortic Insufficiency.—Leonard Hill has shown that the blood-pressure in the arm as compared with that in the leg in normal individuals, at muscular rest, differs only by the hydrostatic pressure of the column of blood which extends from the leg to the arm. In the horizontal posture they are approximately equal. Exercise in the upright posture produces a much wider variation in the leg than in the arm, a fact which is attributed to the mechanism which regulates the pressure to the heart itself and to the vital centers. In patients with aortic insufficiency he has found a constant greater difference between the arm and the leg readings even during muscular quiet in the recumbent posture, so that he considers a marked and constant excess in the systolic blood-pressure of the lower extremities as compared with the upper as pathognomonic of aortic insufficiency. According to H. A. Hare this variation is not present in other valvular lesions. In normal individuals at rest it rarely exceeds 15 mm., while with aortic insufficiency it may amount to 100.

¹ A. E. Taussig and J. E. Cook, *Arch. Int. Med.*, May, 1913, xi, 5.

E. Pesci¹ derives valuable information from a study of the difference in systolic blood-pressure in the brachial

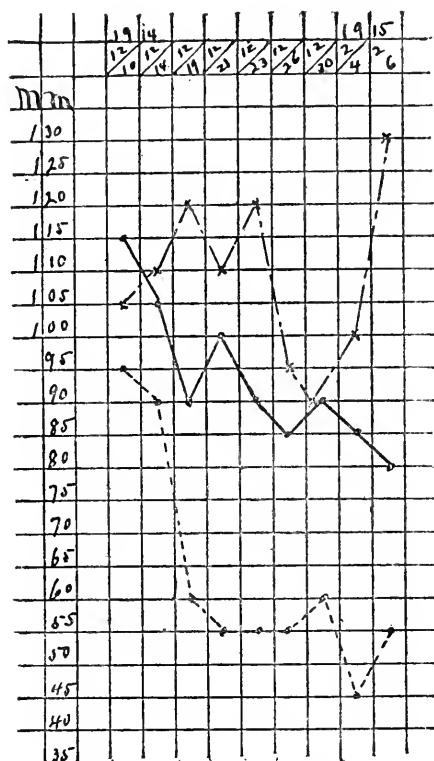


FIG. 67.—Girl. Aged twelve. Mitral valvulitis. Acute endocarditis following follicular tonsillitis. Classical signs of endocarditis with a blowing systolic murmur, slight cyanosis, air hunger and a greatly enlarged and tender liver. The rise in pulse pressure following the institution of treatment, was probably the result of return of more blood to the left side of the heart incident to the relief of pulmonary and hepatic congestion following hot packs. The gradual fall in systolic pressure to termination of the case shows a gradual wearing out of the heart caused by the sudden development of a demand for extra work. The rise in pulse rate is characteristic. The last observation was made shortly before death.

and digital arteries in heart disease. In aortic defects the

¹ *Riforma Medica*, June 7, 1909, xxv, 23.

pressure in both arteries is much higher than in normals, whereas in associated myocarditis the brachial pressure may be high but the peripheral pressure is low. He considers these observations especially instructive when the brachial and digital pressures are determined before and after exercise. If the heart is functionally capable, a normal proportion of pressures between the two readings is maintained. The lower the peripheral reading as compared with the proximal, the weaker the myocardium.

MITRAL DEFECTS

In mitral defects (Fig. 67) and all other compensated valvular lesions, except possibly a mitral stenosis, the blood-pressure is above normal. Starling¹ believes that no matter how ill the patient or how ineffective the work of the heart, the systolic pressure is never below normal. Pesci² states that in mitral defects during the period of compensation the pressure in both the brachial and digital arteries is low, whereas it is above normal when compensation fails (see Fig. 68).

Mitral Stenosis.—The question as to the usual level of systolic pressure in mitral stenosis has not yet been settled. In those cases showing a subnormal pressure it is explained on the ground that the volume of blood actually passing through the heart is sufficiently reduced to cause this low pressure. On the other hand, the belief that the blood-pressure is high in mitral stenosis is based upon the fact that venous pressure is high and the physics of the circulation demand a certain difference between the arterial

¹ *Lancet*, Sept. 29, 1907.

² *Loc. cit.*

and the venous pressures. This point has been dwelt upon by Bishop¹ who states that failure of the circulation in heart disease becomes a matter of concern only when the patient resumes his occupation and even then a low systolic pressure should not be regarded as serious except when it is but little above the venous pressure.

CARDIAC LOAD AND OVERLOAD

Willard J. Stone² has devised a formula whereby the degree of cardiac load and overload may be determined and expressed in comparable figures. This study is based upon the assumption that clinically the pulse pressure represents the load of the heart, which under normal conditions approximates 50 per cent. of the diastolic pressure. The systolic and pulse pressures represent myocardial values, while the diastolic pressure represents arterial resistance and is therefore the arterial factor. The foundation for this contention is based upon a study of sixty-one normal persons whose average pressures were systolic 123, diastolic 80, pulse pressure 40. The amount of energy expended, therefore, to maintain the circulation in excess of that required to open the aortic valve, *i.e.*, overcome peripheral resistance of 80 was 40 (see also my normal chart, page 117). The normal load, therefore, may be considered $\frac{40}{80}$ or 50 per cent. of the diastolic pressure. In twenty-one acute infectious cases that recovered the average pressure was systolic 119, diastolic 76, pulse pressure 42, the load in this case being $\frac{42}{76}$ or 55 per cent. On the other hand in five fatal acute infections the averages

¹ "Heart Disease and Blood-pressure," 1907.

² *Jour. A. M. A.*, Oct. 4, 1913, lxi, 14, p. 1256.

were systolic 102, diastolic 68, pulse pressure 34, and the load $3\frac{1}{6}$ s or 50 per cent.

Preceding circulatory failure, there may be a radical change in the readings, with a tendency for the pulse pressure to equal or exceed the diastolic. Thus in six of fourteen decompensated myocardial cases the pulse pressure approached or exceeded the diastolic. From this it appears that with a load factor of 50 plus an overload of 50 per cent. (*i.e.*, when the pulse pressure equals the diastolic) there is great danger of myocardial failure. Another condition enters here, which is the commonly associated rapid heart rate, which Henderson and Barringer¹ have shown to be a factor. A rapid heart shortens the diastolic period and interferes with ventricular relaxation, thereby preventing the proper filling of the ventricle and in consequence the systolic output is diminished and the mass movement of blood impaired.

Another method of determining myocardial efficiency has been suggested by Masing² who notes the effect of exercise upon the relation of systolic to diastolic pressure. After exercise a normal circulatory apparatus will yield a systolic rise of greater extent than the diastolic rise, *i.e.*, the pulse pressure is increased, while after exercise a defective myocardium may show the rise in both pressures, but they will tend to approximate and consequently the pulse pressure will be reduced. In my experience this change cannot always be demonstrated, although in certain cases it is undoubtedly so.

¹ Y. Henderson and T. B. Barringer, *Am. Jour. Physiol.*, 1913, xxxi, p. 288.

² E. Masing, *Deutsch. arch. f. klin. Med.*, 1902, lxxiv.

Graphically we may express the change as follows:

	Normal myocardium		Defective myocardium
Before exercise . .	$\frac{\text{P.P.}}{\text{S.P.}} = \frac{35}{135} = \frac{1}{4}$		$\frac{\text{P.P.}}{\text{S.P.}} = \frac{40}{140} = \frac{1}{3}$
After exercise . . .	$\frac{\text{P.P.}}{\text{S.P.}} = \frac{55}{175} = \frac{1}{3}$		$\frac{\text{P.P.}}{\text{S.P.}} = \frac{30}{135} = \frac{1}{5}$

In this the increase in the value of the fraction is an indication of efficient heart, while the decrease in the value of the fraction after exercise is an indication of inefficiency.

In my own practice I have derived much satisfaction from a computation, first suggested by Gibson, in which an effort is made to demonstrate the relative work and velocity factors of the cardiac energy, modified to meet the conditions met in auscultatory determinations.¹ This is based upon the following propositions: First, the normal systolic, diastolic, pulse pressure relation is 3 : 2 : 1; and second, if the pulse pressure represents the systolic output (see page 137) it follows that it must also be the most important factor in determining the velocity of the blood-stream, which for physical reasons must bear a definite relation to the volume output of the heart and to the caliber of the conduits—the arteries—so that, if other factors remain the same, it is not a difficult matter to estimate both the velocity and also the work of the heart while operating under either normal or abnormal conditions.

The above propositions can be arranged graphically as follows.² For example, take a case presenting these figures: S.P. 130, D.P. 85, P.P. 45, P.R. 70; then — P.P. (45) \times P.R. (70) = Velocity (3100), and S.P. (130) \times P.R. (70) = Work (9100).

We may carry our calculations further and state that the

¹ *Interstate Med. Jour.*, p. 897, xxi, 8, July, 1914.

² F. A. Faught, *Interstate Medical Journal*, July, 1914.

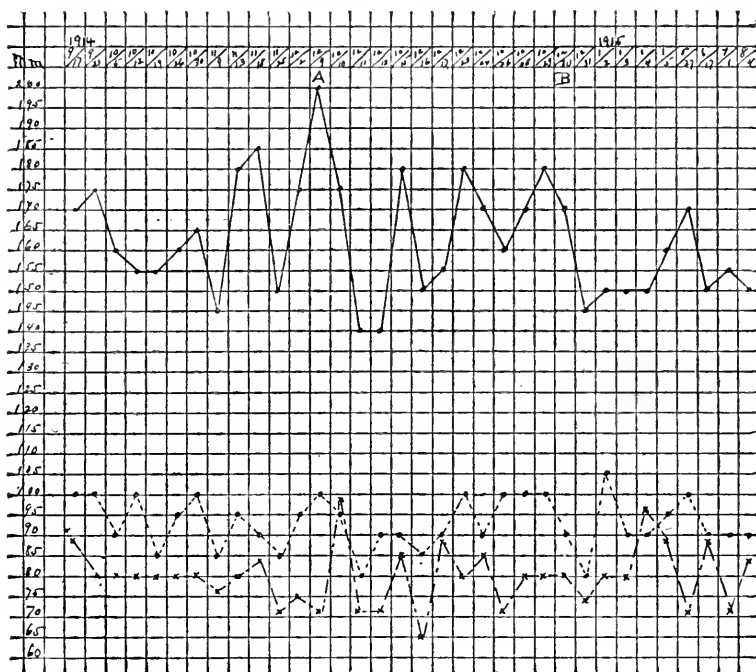


FIG. 69.—Female. Aged sixty. Typical cardiac neurasthenic. Has been in the hands of many physicians during the past ten years with indifferent results. Complains of a long list of vague and variable symptoms including insomnia, joint pains, globus hystericus, dyspnea, heat-flashes, palpitation, variable appetite and nervousness.

Physical examination shows a moderate degree of chronic myocarditis which is well shown in the chart by the abnormally large pulse pressure. At no time during observations did this patient show any marked signs of myocardial weakness, the condition being more one of instability.

At (A) a sharp rise in systolic and pulse pressure was accompanied by a number of dizzy spells and a tendency to faintness, the cause for which was unexplained. A prompt fall in pressure resulted from an administration of atropin with strychnin, followed by effect obtained at (B), due to better hygiene and systematic out-door exercise. This was the only time during the observation of this patient that the symptoms were suggestive of myocardial overstrain. This is the type of case which, on account of extreme personal solitude, and the avoidance of all unnecessary strain that would tend to upset a circulatory equilibrium will go on for years and may live to extreme old age in spite of the cardiac-muscle handicap.

Fair cardiac efficiency is indicated by the regularity with which the pulse pressure follows the systolic.

velocity and the work, as estimated by the above formula, also bear a definite normal relation which is dependent entirely upon the normal relation of pulse pressure to systolic pressure (and that this relation is as 3:1, while in myocardial cases the ratio is increased).

Application.—Case of chronic myocarditis and arteriosclerosis.

Before treatment:

S.P. 210, D.P. 100, P.P. 110, P.R. 104.

$$\left. \begin{array}{l} \text{S.P. (210)} \times \text{P.R. (104)} = \text{Work (21,840)} \\ \text{P.P. (110)} \times \text{P.R. (104)} = \text{Velocity (11,440)} \end{array} \right\} = \text{ratio 1:2.}$$

After two weeks' treatment:

S.P. 195, D.P. 140, P.P. 55, P.R. 84.

$$\left. \begin{array}{l} \text{S.P. (195)} \times \text{P.R. (84)} = \text{Work (14,580)} \\ \text{P.P. (55)} \times \text{P.R. (84)} = \text{Velocity (4,620)} \end{array} \right\} = \text{ratio 1:3.}$$

Here under the proper method of treatment the work velocity was greatly benefited, the actual work reduced one-third, so that while the heart was at first barely able to maintain the needs of the case under serious strain, accompanied by evident signs of cardiac distress, after two weeks the danger of acute failure of the circulation was overcome and the whole complexion of the case altered for the better.

Importance of Diastolic Pressure.—From his study Stone concludes that since the diastolic pressure measures the peripheral resistance, it is a better index of hypertension than is the systolic pressure. The diastolic pressure is less influenced by pathologic factors than the pulse pressure.

Pulse Pressure in Cardiac Neuroses.—Stone believes that marked variations of pulse pressure occurring at

short intervals (Fig. 66) in cases with cardiac symptoms, are very suggestive of a cardiac necrosis.

Blood-pressure in Auricular Fibrillation.—Silverberg¹ studied eight patients all of whom were typical examples of this variety of cardiac irregularity, all of which showed the wide range of blood-pressure which an individual case of auricular fibrillation may possess. He found that the blood-pressure reading of the small beats varied from 80 to 160, while the maximum systolic pressure varied between 200 and 210.

Paroxysmal Tachycardia.—The pulse rate may be from 150 to 300, the heart sounds good and the pulse small, sometimes the pulse rate cannot be counted, the blood-pressure is usually found to be low, probably because the shortness of diastole does not allow the proper filling of the ventricles; the venous pressure is high. In the intervals the circulation is apparently normal (Krehl).

Bradycardia.—The effect on blood-pressure is variable, depending on the cause and on other conditions if present. When extreme, blood-pressure is always lowered; patients with dyspnea cannot exert themselves, and even change in posture may precipitate attacks of syncope (Krehl).

Finally, in close relation to the circulation in diseases of the heart, as has been found by Krehl and others, are the last stages of arteriosclerosis. Here there is widespread dilatation of the splanchnic area together with failure of the heart to respond to the demands made upon it; this results in a gradually falling blood-pressure, when therapeutic measures have little or no effect (see Terminal Hypertension).

¹ *Brit. Med. Jour.*, Apr. 6, 1912, i, 2675.

Cardiac Asthma.—This term implies a severe attack of dyspnea occurring in an individual having heart disease. During the attack the pulse is rapid, soft and irregular in force and rhythm. The blood-pressure is usually below normal during the height of the attack, speedily regaining its former level as the attack subsides.

CHAPTER XX

RELATION OF BLOOD-PRESSURE TO SURGERY

The surgeon has not been as rapid in the adoption of blood-pressure studies to his pathologic problems as has the general physician, neither does it appear that the two stand upon the same ground in regard to the significance of the findings; thus many medical men view with alarm abnormalities in blood-pressure, particularly the persistent elevation in the systolic, when found in cases demanding operation, whereas the surgeon is inclined to doubt the truth of this opinion, so that he often underestimates the value of preliminary blood-pressure examinations in surgical cases. For example, the active practitioner of medicine as a matter of course includes a series of blood-pressure observations in his general survey of the case and often depends largely upon its indications to determine the condition of the kidneys and circulation in relation to the risk of operation, especially in determining for and against operations of choice (*i.e.*, not urgently demanded). On the other hand many surgeons, some of wide reputation, do not study blood-pressure at all, even in cases evidently hazardous. This is a most unfortunate attitude, and in the present state of our knowledge of the subject hardly tenable. It has been shown beyond a question of doubt that a knowledge of blood-pressure before, during and after surgical procedures, especially when they are prolonged

and trauma is great; when the individual is obviously in a poor condition and predisposed thereby to shock, is of definite value, and its estimation may assist materially in preventing untoward results. Even admitting the tendency in any new subject to go to extremes, it is evident that there must be a middle ground which, when occupied by the surgeon, will vindicate this test in connection with surgical procedures.

That the surgeon is gradually turning to a consideration of the sphygmomanometer and its findings is shown by the recent surgical literature upon shock, which includes a study of the relation of blood-pressure to this condition,

In spite of this the urinary excretion fell to 7 ounces in twenty-four hours and was reported as resembling that in eclampsia. The systolic pressure was 120 and the pulse pressure in the next twenty-four hours fell to 20, indicating a myocardial and not a nephritic involvement. At this point hot packs and sweating would undoubtedly have occasioned a fatal termination, whereas active hypodermic stimulation by adrenalin and caffein resulted in a return of cardiac equilibrium, an increase in kidney output and a normal progress until (C), when several hours before the observation recorded, the patient became suddenly pale and had profuse perspiration with an imperceptible pulse. Oxygen and hypodermic treatment tided her over this crisis and a thorough examination at the time of the blood-pressure test (C), failed to show that this attack had any effect upon the patient's convalescence. Subsequent investigation showed that the attack was probably one of ptomain poisoning, as the patient had eaten fish for the first time on that day, and was reported as having had several attacks in the past from the same cause.

Several points stand out significantly in this case:

First.—The beneficial effect of eliminative measures preliminary to operation in cases of evident renal involvement.

Second.—The information gained by blood-pressure test immediately after operation which was the only means of determining the true cause of the grave symptoms shown.

Third.—The fact that such a cardiovascular system can withstand the effect and shock of prolonged operation without suffering apparent damage as shown by the subsequent records.

Fourth.—The mental relief afforded those in charge by eliminating the possibility of embolism as the cause of the attack occurring at (C).

and upon which all contending factions admit the definite and often intimate relation between blood-pressure and shock, whether as cause or effect.

A single example which recently came to the writer's attention may serve to demonstrate that there are times when the surgeon *must* employ the blood-pressure test, and when such study will contribute largely to the successful outcome of the case (Fig. 70). A woman of about forty-five, suffering from the effect of an old pelvic inflammatory condition, was found to have a moderate degree of contracted kidney as shown by repeated urinalysis, and the blood-pressure, which ranged between 180 and 200 systolic. Operation was indicated most urgently on account of intense, prolonged and frequent attacks of pain, rendering the patient a semi-invalid. Preliminary preparation of a dietetic and hygienic nature showed, on the day before the operation, a phthalein output of 50 per cent., first hour, with 20 per cent. second hour, a copious urinary excretion and a blood-pressure of systolic 170, diastolic 110, pulse pressure 60. Many adhesions occasioned a prolonged and difficult operation, followed by some degree of shock, so that during the first succeeding twenty-four hours the urinary output was 7 ounces, and was reported on examination to be like that found in eclampsia, suggesting the supervention of uremia.

A blood-pressure determination gave the following: systolic 120, diastolic 100, pulse pressure 20, pulse rate 104, with an absence of the second and third auscultatory phenomena over the brachial; these together with the low urinary excretion, pointing to a cardiovascular and *not* a nephritic condition. The prompt intravenous use of

adrenalin, and the hypodermic administration of caffein and digitalis resulted in a prompt rise in systolic and an increase in pulse pressure, with a return of the second and third (cardiac strength) sounds. These changes were coincident with a marked increase in urine output, and abatement of the pathologic urinary findings, so that on the third day the blood-pressure record was systolic 130, diastolic 85, pulse pressure 45, pulse rate 92, while on the fifth day the urine was over 40 ounces in twenty-four hours, almost normal by the laboratory report and the patient in most excellent condition.

The points to be emphasized here are:

1. The chronic nephritis prior to operation.
2. The normal phthalein output.
3. The scant, eclamptic urine with great circulatory depression, the latter being the only definite indication that the difficulty was not uremic but cardiovascular, and finally the prompt response following treatment from the latter standpoint only.

Clinical and experimental study has demonstrated many facts in connection with the relation of blood-pressure variations to the various surgical procedures, incidents and accidents. Possibly the reason for the failure of the surgical fraternity to adopt and interpret blood-pressure findings is because they have so far failed to thoroughly study this relation. Continued study of routine blood-pressure records will eventually force the conclusion that there is often a definite relation between the height of the blood-pressure and variations in its several factors, to the general clinical picture, so that a proper interpretation of blood-pressure in relation to other symptoms may

become of signal value in following or in predicting post-operative complications.

It is no argument against the employment of this test that we do not yet know precisely the nature, causes and mechanics of shock; it rather enhances its value in that it throws another safeguard around the extreme case.

That this point is beginning to be appreciated is shown by the writing of at least one well-known surgeon¹ who states that when the systolic blood-pressure falls to 100 or lower it is time to consider emergency measures, and that often in the absence of any other signs of impending shock there is a more or less rapidly falling systolic pressure; also he believes that many cases of collapse following the removal of the patient from the operating room may be avoided by the comparison of the observations of blood-pressure taken before and after operation.

It has also been shown that when primarily the blood-pressure is abnormally high or abnormally low, complications are more apt to ensue and that one should be on guard against thrombosis, embolism, pneumonia, nephritis, and bronchitis² under such conditions.

In the average surgical case, lethal accidents are either the result of acidosis or the paralyzing effect of the anesthetic upon the cardiac or the respiratory centers, so that in prolonged operations the study of the blood-pressure during the operation may be a valuable guide in indicating the reaction of the patient while under the anesthetic. In other words the persistence of a fair average systolic level and an adequate pulse pressure is indicative of a

¹ Joseph C. Bloodgood, *Penna. Med. Jour.*, January, 1912, p. 256.

² G. W. Crile, *Jour. A. M. A.*, May 3, 1913, lx, No. 22, p. 1737.

better factor of safety than either a sudden rise, or gradual or marked fall in systolic pressure, or a diminution in the pulse pressure.

The employment of the sphygmomanometer would seem to have placed the administration of anesthetics upon a firmer foundation since, while our judgment in regard to the condition of the patient under operation may be modified by a knowledge of the pathologic condition leading up to the operation and by the general physical condition of the patient at the beginning of the operation, nevertheless it has been borne out by clinical experience that the blood-pressure test is of value as an indication of safety during prolonged and extreme operations, and it is hoped that in a short time the careful surgeon will show a greater tendency to the routine of employment of this test, and by applying the knowledge which we now possess in relation thereto, add further to the enlightenment of this important subject.

APPLICATION OF THE TEST TO SURGICAL CASES

Blood-pressure observations can usually be made by the anesthetist, although the undivided attention of an assistant, either a nurse or student trained to make these observations, should be used whenever possible. Observations made during surgical operations should occur at from two- to five-minute intervals while an expert assistant can give reports of blood-pressure once every minute.

In grave cases the value of the test becomes greater as the interval of observation is shortened, for it is possible for serious changes in the circulation to occur in a very short space of time. The observation to be of greatest service

should be charted and kept in view of the surgeon. Changes in the auscultatory phenomena should also be reported to the surgeon and to the anesthetist. The value of these observations lies not only in the facility with which dangerous alterations in blood-pressure may be detected, but also in the fact that the effect of restorative and stimulating measures may be noted, so that efficient dosage may be employed. A falling systolic pressure with a rising pulse rate is an indication for immediate action, while a diminution in pulse pressure, if progressive, is an indication of a failing circulation, usually attributable to the myocardium.

In the study of blood-pressure under anesthetics it is necessary to obtain the patient's normal systolic pressure before the anesthesia is begun, and this should, if possible, be obtained the day before, or at least previous to the patient's final preparation and appearance in the operating room. Observations made immediately before anesthesia will frequently show an abnormally high pressure and an accelerated pulse rate, which is accounted for by the stimulating effect of excitement and fright on the cardiomotor and vasomotor centers.

It must also be borne in mind that the blood-pressure level will be affected by rest in bed, and by restricted diet which usually precedes surgical operations, so that due allowance should be made for these influences.

In order to intelligently interpret the results of sphygmanometry the surgeon should know the effect of the ordinary steps of surgical procedure upon blood-pressure as compared with the extraordinary and dangerous manifestations resulting from excessive trauma, hemorrhage, etc.

Pain causes a temporary rise in blood-pressure. Abdominal pain, in which the splanchnic nerves are involved, greatly increases the pressure on account of the resulting constriction of the splanchnic vessels. H. Curschmann¹ believes that by the sphygmomanometer we may be able to differentiate between the several sources of abdominal pain, and cites instances where the pain from gastric and intestinal crises, as in tabes and in lead colic, caused the pressure to run up to 170 to 200 mm., to drop again to normal as soon as the pain ceased, whereas in pain from gastric ulcer, gall-stones and appendicitis there was only a moderate increase.

Fear.—As fear, apprehension and mental excitement are usually accompanied by a sudden increase in pulse rate, so the systolic pressure tends to rise from 10 to 20 mm. above the average. If the element of fear is sufficiently profound to induce shock, there may be a fall in systolic pressure amounting to 60 mm. in a few minutes.²

INFLUENCE OF OPERATIVE PROCEDURES

Skin Incision.—Incision of the skin, except in profound anesthesia, results in a reflex vasoconstriction which shows itself by slight rise in systolic pressure, amounting ordinarily to from 5 to 10 mm. Hg.

Incision of the Peritoneum.—This procedure causes a sudden and sharp rise in systolic pressure, which, though momentary, is more marked than that when the skin is incised; extensive trauma of the peritoneum is prone to cause a marked systolic fall, the degree and extent of de-

¹ *Münch. med. Wochen.*, Oct. 16, 1907.

² Bloodgood, *Internat. Jour. Surg.*, September, 1913, xxvi.

pression depending upon the extent and degree of trauma and exposure to which the viscera are subjected (degree of shock).

Aspiration of Fluid from the Chest and Abdomen.—Capps¹ and Lewis² have analyzed the results of extensive observations of the blood-pressure before, during and after paracentesis and have classed them under two heads.

(a) *Constant Features.*—Following the transient rise incident to spine puncture or incision, there is, during drainage of fluid, a marked fall in arterial pressure, averaging 32 mm.; the depth of depression is usually reached after the needle has been withdrawn, although it may occur at any time. The greatest fall noted by these observers was 62 mm., which followed the removal of 2000 c.c. of fluid. Occasionally the fall is very slight. The final pressure taken an hour after the end of the operation averages a fall of 12 mm. This decrease corresponds to an average fall in intra-abdominal pressure, after tapping, of 10 to 14 mm. as noted by Querin.

(b) *Special Influences.*—The rate of withdrawal of fluid, rather than the amount, appears to be the important factor influencing systolic pressure, being greater the more rapid the withdrawal. External pressure upon the abdominal wall after drainage reduces the final total fall by from 5 to 20 mm. Posture is important, as normally the pressure is higher when sitting than when recumbent. At the end of paracentesis a change from the sitting to the prone position increases the pressure 6 to 20 mm. and this rise is more marked in those cases which suffer the most

¹ *Jour. A. M. A.*, xlviii, 1, 1907.

² *Am. Jour. Med. Sci.*, December, 1908.

from the effects of the operation. These changes are also noted during the removal of purulent effusions. Alarming symptoms appear more frequently during the removal of pleural effusions. Capps and Lewis believe that this blood-pressure change is not purely the result of alteration in intrathoracic pressure but is due to the activity of two reflexes, one a cardio-inhibitory and the other a vasodilator. It is believed that the effect of these reflexes can be materially modified if sufficient care is exercised in the introduction of the needle, so as to avoid wounding the visceral pleura.

Induced Pneumothorax.—F. C. Smith¹ has studied the influence of induced pneumothorax on the blood-pressure in sixteen cases in which the effect of the operation itself was excluded by taking the pressure at other times and extending them over varying periods up to more than a year after the operation. No important changes in blood-pressure were noted.

Gynecologic Operations.—Reliable observations show that manipulations of the pelvic organs cause a rise in systolic pressure and that this rise is proportionate to the severity of the traumata, unless shock intervenes. The reports of observers employing chloroform as an anesthetic are of little value because of the uniform depressing effect of the chloroform itself.

Cord and Brain Operations.—Operative procedures for decompression are attended with added danger, because of the primary lowering of systolic pressure which follows the escape of cerebrospinal fluid.² The amount of subsequent

¹ *Jour. A. M. A.*, May 29, 1915, lxiv, 22.

² E. D. Fisher, *Jour. A. M. A.*, Aug. 3, 1912, lix, 5, p. 395.

circulatory depression will depend upon the nature, duration and extent of the operative procedures and the degree of the trauma or the extent of the disease preceding operation. It is stated by Crile that, under normal conditions, dural incision *per se* has little if any effect upon the systolic blood-pressure curve, but that irritation of the brain or cord covering by sponging, packing, etc., is usually occasioned by a sharp fall in systolic pressure.

Lumbar Puncture.—After the primary rise coincident with skin puncture which occurs in the conscious patient, the withdrawal of fluid tends to reduce systolic pressure, but the net result of lumbar puncture is a rise, averaging 20 mm. for at least twenty minutes succeeding the removal.¹

The type of blood-pressure curve in lumbar puncture in individuals with increased intracranial tension of supratentorial origin is the same as in normal individuals, while in subtentorial cases the primary rise is more sustained.

The blood-pressure curve in cases subjected to high spinal anesthesia shows a primary sharp rise accompanying and immediately following the puncture, after which, as the anesthetic effect upon the thoracic muscles occurs, there is a fall, which is not lost until the paralyzing effects of the drug have passed off.

Geo. G. Smith² has noted a fall of as much as 100 mm. Hg., after the induction of high spinal anesthesia. In several cases the systolic level fell to 45 mm. Hg. These extremely low pressures occurred only in a small proportion of spinal anesthetics; in many instances the systolic

¹ H. T. Gray and L. Parsons, *Quart. Jour. Med.*, April, 1912, 9.

² *Boston Med. and Surg. Jour.*, Sept. 30, 1915, clxxiii, 141, p. 502.

pressure fell to 75 mm. Hg. Many of the cases showed a marked reduction, exhibiting signs of shock. The pulse usually remained between 60 and 90, and did not increase in rate as the blood-pressure fell. These low pressures usually appeared in from ten to twenty minutes after the introduction of the drug, and remained for from ten to fifteen minutes after the action of the drug had passed off.

Smith summarizes our knowledge of the effects of spinal anesthesia by the usual anesthetics as follows:

1. The blood-pressure may be lowered as much by spinal anesthesia as by section of the cord in the cervical region.

2. The greatest fall follows injection of the anesthetic in the thoracic region, where the vasomotor fibers which supply the splanchnic area leave the cord.

3. This part of the vasomotor mechanism is the first to be affected by the drug when the injection is made in the lumbar region.

4. The extent of diffusion of the drug within the dural sac appears to be influenced more by the bulk of the solution injected than by any other factor.

5. A given amount of drug in a concentrated solution diffuses less, and consequently lowers blood-pressure less, than does the same amount of drug in a more dilute solution.

6. The greatest effect upon blood-pressure is noted when adrenalin is combined with novocain.

7. Entire fixation of the drug does not take place within twenty minutes after the time of injection.

8. When a solution of greater specific gravity than spinal fluid is used, the extent of diffusion may be increased by gravity.

9. Attempts to raise the blood-pressure by the intraven-

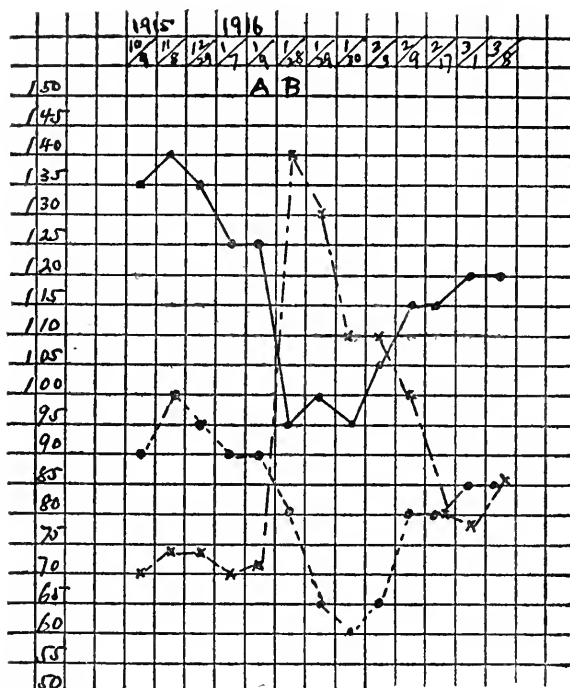


FIG. 71.—Male. Aged forty-two. Hemorrhage from gastric ulcer. Examination prior to hemorrhage (A) shows a slightly elevated systolic pressure in one of rather active physical and mental habits. Pressure recorded at (B) was made a few hours after a copious hemorrhage from gastric ulcer, at which time the patient was almost totally blind, showed extreme pallor and apathy, with a rapid-running pulse, and hurried gasping respiration. By actual measurement the amount of blood lost within an hour or two was not less than a quart. This is the characteristic curve of massive hemorrhage in which the greatest reduction first occurs in the systolic pressure resulting in an extremely small pulse pressure immediately following. Circulatory balance is rapidly established by a reduction in diastolic pressure with or without much change in systolic pressure, resulting in a rapid return to a safe pulse pressure usually several days before the systolic pressure again approaches normal.

ous injection of pituitrin or adrenalin during spinal anesthesia give only a small and very transitory rise.

10. Adrenalin alone injected into the spinal canal was found to have no effect on blood-pressure.

Hemorrhage.—The amount of reduction in systolic pressure caused by hemorrhage as a rule bears a direct relation to the amount of blood lost and to the rapidity with which the loss of blood occurs. An exception to this is seen in cerebral hemorrhage where increased intracranial pressure occasions great elevation.

John B. Briggs¹ has reported intracranial hemorrhage with a systolic pressure of 400 mm. Hg. In the usual acute hemorrhage, as from external wounds, from gastric ulcer or during typhoid fever, in tuberculosis and in epistaxis, the systolic pressure may fall so low as to endanger life. This complication becomes more serious when the hemorrhage is engrafted upon a weakened state caused by previous hemorrhage, or during collapse when the vasomotor system is greatly weakened or paralyzed. It is noteworthy that the fall, even when marked, is transient unless the hemorrhage continues, so that the value of this sign diminishes in proportion as the time between the hemorrhage and the observation is prolonged (Fig. 71). It is fortunate that all hemorrhages, except those coming from very large vessels, tend to become arrested long before great danger due directly to loss of blood occurs. The fall of pressure from hemorrhage is not as great as the actual amount of blood lost would indicate; this is due to the reflex augmentation of cardiac output and also to a rapid drawing upon the fluid in the lymphatic system. If arrest

¹ *Johns Hopkins Hosp. Bulletin*, Aug. 10, 1910.

of hemorrhage fails to occur before these reserves are exhausted, then the circulation fails, because the mechanical function of the heart cannot be carried out (see also shock). In other words the amplitude of cardiac contraction decreases chiefly because of the lack of sufficient fluid in the actual system upon which to operate. Henderson and Barringer¹ ascribe this result to the evident fact that the venous pressure is greatly lowered, and conclude that the so-called "critical factor" in hemorrhage is dependent largely upon a failure of venous supply to the right heart, which results in failure of the circulation as a whole. On the other hand, it may be that lowered venous pressure decreases the heart output indirectly by lessening pulse-pressure on the arterial side, thereby reducing the rate and volume of actual flow, which, being inadequate to maintain the nutrition of the important centers, contributes to the fatal termination.

Diagnosis of Concealed Hemorrhage.—Carl J. Wiggers² recommends a frequent determination of the pulse-pressures in cases of suspected internal hemorrhage, believing that frequent estimation of the blood-pressure in suspected hemorrhage is of great value in differentiating this complication from others also accompanied by a falling pressure. He has found that almost invariably a progressive decrease in pulse-pressure accompanied by an acceleration in pulse-rate, occurring after surgical procedures, is indicative of continued bleeding and that the converse, if persistent after several observations, indicates a cessation of the bleeding. He recommends that the following series of

¹ *Loc. cit.*

² *Arch. Int. Med.*, September, 1910 and *Arch. Int. Med.*, July, 1914

diagnostic features be considered in cases of suspected or evident internal hemorrhage.

In arrested hemorrhage the pulse pressure tends to be abnormally large. If the respiration undergoes little or no change, the following deductions may be drawn: (a) A progressive decrease in pulse pressure with a decrease in the product of the pulse pressure and the pulse rate, indicates a continuance of the bleeding. (b) An increase in both, if permanent, after several determinations, indicates a cessation of hemorrhage. (c) A temporary increase of both followed by a marked decrease as shown by subsequent examinations, indicates an exacerbation. These points have been experimentally proven by animal research.

In all operations control of hemorrhage is an important factor in maintaining blood-pressure. When hemorrhage is slight and well controlled the effect on pressure is usually unimportant and does not call for special treatment. On the other hand, operations accompanied by considerable bleeding may result in severe and dangerous hypotension. The tendency to shock is greatly increased by hypotension from any cause during anesthesia, but if shock is successfully combated, pressure soon returns to a safe level.

INFLUENCE OF ANESTHETICS ON BLOOD-PRESSURE

Discussing the mechanism of the action of anesthetics on blood-pressure Guy, Goodall and Reid¹ remark that blood-pressure may be *lowered* by (1) depression of the heart; (a) by vagus inhibition, either by direct stimulation of center by the drug, or by reflex stimulation through the nervous system; (b) by weakening of the heart muscle.

¹ *Edinburgh Med. Jour.*, August, 1911.

(2) Dilatation of the vessel wall or paralysis of vasomotor tone. Blood-pressure may be *elevated* by (1) stimulation of the heart; (a) by excitement; (b) by stimulation of the heart by the drug. (2) Stimulation of the vasomotor centers; (a) by the action of the drug; (b) by asphyxia.

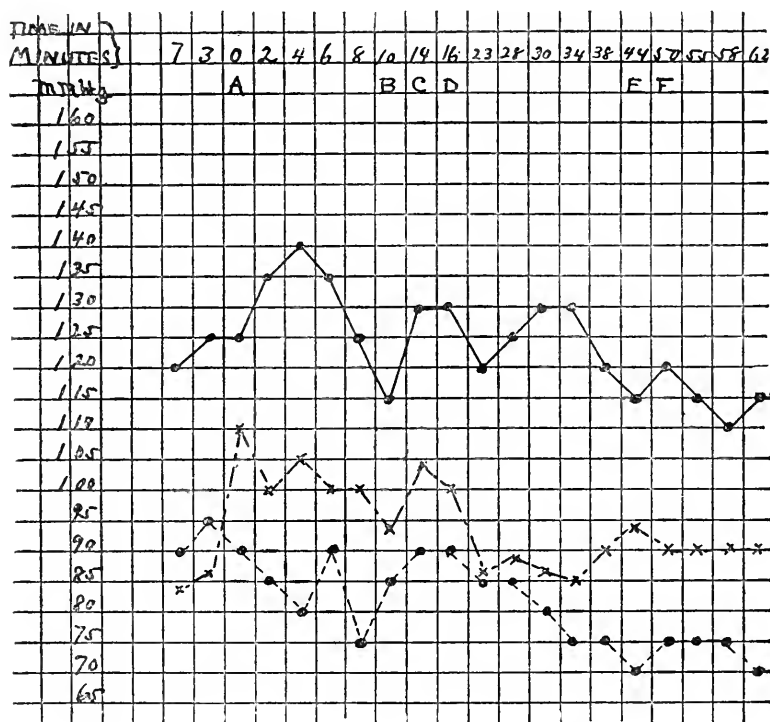


FIG. 72.—Ether anesthesia—laparotomy. Aged twenty-three. (A) Anesthetic begun. (B) Anesthesia complete. (C) Skin incision. (D) Peritoneal cavity opened. (E) Peritoneum closed. (F) Anesthesia discontinued.

Experiment and clinical study show that the different anesthetics in general use affect the circulation and blood-pressure in different ways, and that the extent of the depressing effect of the anesthetic on blood-pressure de-

termines in a great measure the relative danger of that anesthetic and its tendency to produce shock.

In the following paragraphs an effort has been made to indicate as far as is known the action of different anesthetics

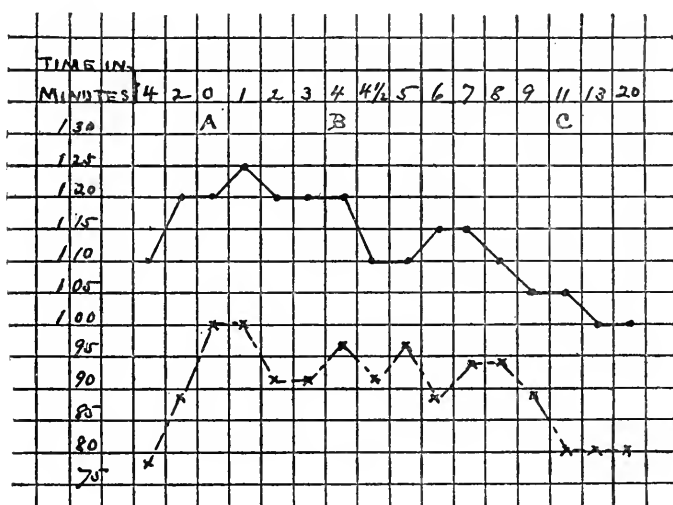


FIG. 73.—Anesthetics. Experimental use of chloroform in a male aged thirty-two. Complete anesthesia induced. Anesthetic begun at (A), complete at (B), and terminated at (C). Duration of anesthesia eleven minutes. Note the absence of usual rise in pulse rate and systolic pressure incident to mental excitement before the administration of the anesthetic, the sharp rise in pulse rate at the onset and the steady and rather marked fall in systolic pressure and pulse rate throughout the administration together with a return to approximately normal pulse as the case regained consciousness.

and to show what blood-pressure changes may be expected to occur under them.

Ether.—Ether does not occasion any great alteration in blood-pressure. The administration of ether may cause a primary rise or a fall, or it may maintain, as in most cases, a constant level (see Fig. 72). It causes more rapid and more forcible cardiac action, with dilatation of the smaller

vessels, the latter effect probably counteracting the former, thereby contributing to the maintenance of a constant blood-pressure level. The development of shock under ether is accompanied by great systolic depression from which subsequent recovery is slow.¹

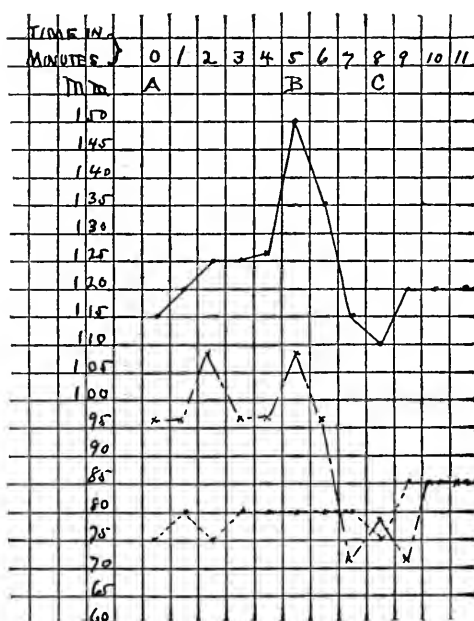


FIG. 74.—Nitrous oxide anesthesia. Male. Aged thirty-one years. Note rapid and sharp rise in all pressures incident to the administration of the drug (A to B) and rapid fall to a point approximating the initial pressures at the end of the administration. The great increase in pulse pressures here is largely due to the sharp rise in systolic pressure as the diastolic remained practically stationary throughout the administration. This is a condition which is characteristically met in nearly all cases of nitrous oxide anesthesia.

Chloroform.—Almost without exception, chloroform causes a reduction in blood-pressure amounting to 10 or 20 mm. (see Fig. 73), the degree of depression depending

¹ H. P. Fairlie, *Lancet*, Feb. 28, 1914, i, No. 4722.

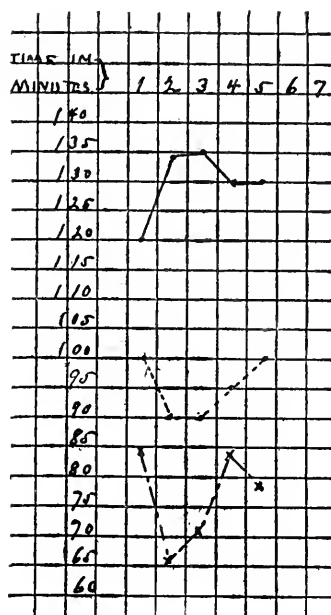


FIG. 75.—Nitrous oxide anesthesia. Young adult. Extraction. Duration of administration four minutes. Note extra strain thrown upon heart muscle as shown by over 50 per cent. increase in pulse pressure. Also marked depression in pulse rate resulting from automatic effort of cardio-motor center to minimize the systolic rise.

	BEFORE			DURING			AFTER		
	MAX	MIN	AV	MAX	MIN	AV	MAX	MIN	AV
Systolic	165	110	133	172	115	134	140	110	130
Diastolic	110	70	92	115	80	96	110	80	96
Pulse Rate	144	60	78	144	60	89	96	60	76

FIG. 76.—Summary of nitrous oxide-oxygen anesthetics by Harley Stamp. Sixteen administrations of from two and one-half to seven minutes' duration in subjects between fourteen and forty-eight years. These were all normal as far as their cardiovascular system was concerned and none of them suffered any ill effects from the administration, despite the great variations in all readings occasioned by the gas.

upon the concentration of the drug. The fall may occur suddenly and be dangerous even after the administration of only a few cubic centimeters (Fairlie).

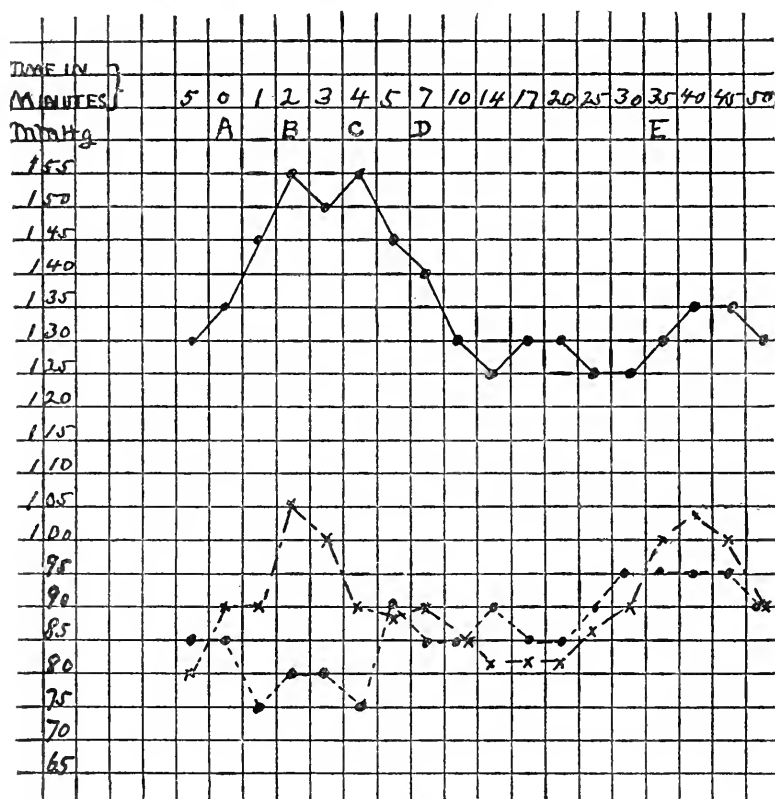


FIG. 77.—Nitrous oxide-ether anesthesia. (A) Anesthetic begun. (B) Nitrous oxide anesthesia complete and ether begun. (C) Patient struggling. (D) Anesthesia complete. (E) Anesthetic discontinued. Note the rapid rise from (A) to (B) incident to partial asphyxia of nitrous oxide and the second rise (C) incident to muscular activity.

Chloroform is dangerous in all stages of its administration, the greatest danger being at the beginning of the administration. Struggling by the patient seems to increase the bad effect.

Nitrous Oxid.—Nitrous oxid, when given alone in sufficient concentration to induce rapid anesthesia, usually causes a short elevation in blood-pressure, due to the partial asphyxia induced (Figs. 74, 75, and 76). This rise is not so marked when rebreathing is allowed (Guy, Goodall and Reid) and is almost entirely eliminated when a gallon

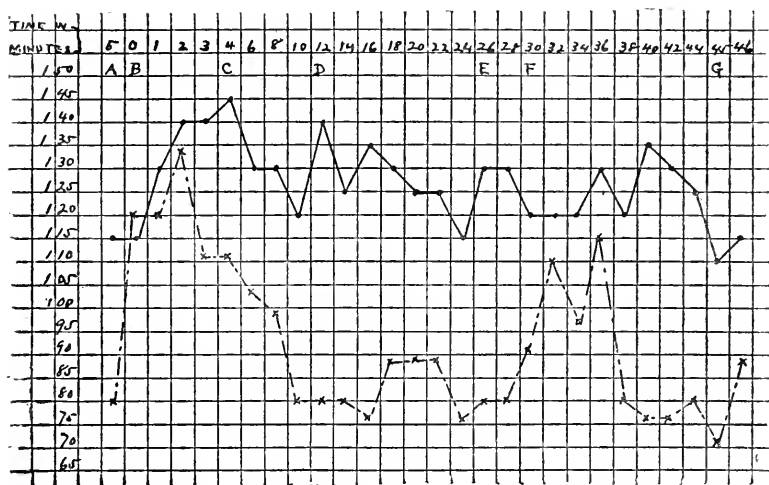


FIG. 78.—Nitrous oxid-oxygen anesthesia by Dr. W. H. Smith. Male. Aged forty-five. Operation double herniotomy. (A) Preliminary observation. (B) Anesthetic begun. (C) Right-side incision. (D) Marked cyanosis relieved by increase in oxygen percentage. (E) Left incision begun. (F) 10 per cent. ether vapor added for two minutes on account of cyanosis and increased reflex action. (G) Operation completed. Duration of anesthetic forty-six minutes. Relaxation fair throughout except at (D) and (F). Patient's condition good at all times in spite of marked pallor and continued profuse perspiration.

of oxygen is inhaled first, although the employment of oxygen in this way curtails by a few seconds the available period of anesthesia. This is a point of value in cases of essential hypertension.

Nitrous Oxid-Ether Sequence.—This anesthetic causes a gradual elevation of pressure, until the stage of complete

anesthesia is reached, when it has practically the same effect as ether anesthesia (Fig. 77), while the induction of shock in susceptible cases seems to be greatly delayed.

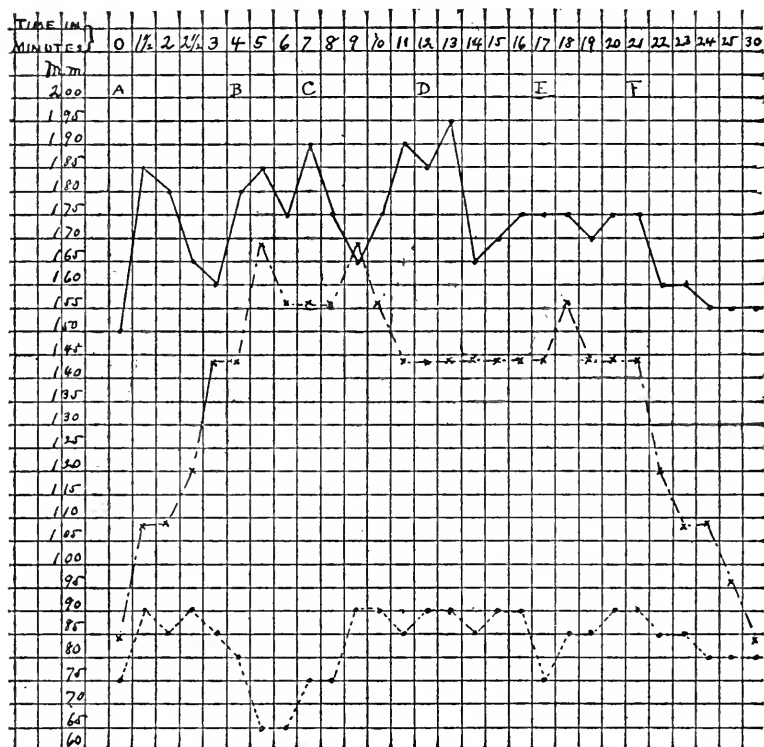


FIG. 79.—Ethyl chlorid anesthesia. Male. Aged sixteen. Removal of tumor of lower jaw. Duration of anesthesia twenty-two minutes. Amount of anesthetic used 12 c.c. (?). The important feature of this chart is the profound effect of this drug upon the pulse rate, and a decided initial rise in systolic pressure, with an exacerbation incident to the breaking of each fresh tube as shown at A, B, C and D. A rather irregular rise in pulse pressure incident to a variable systolic pressure, with a return to approximately normal values within a few minutes after the end of the operation. (A) Anesthetic begun, first tube. (B) Second tube. (C) Third tube. (D) Fourth tube. (E) Sutures begun. (F) Anesthetic withdrawn.

Nitrous oxid combined with oxygen produces a primary rise in blood-pressure, which immediately falls to normal,

as the state of analgesia is reached. The proper control of this tendency to elevation by the oxygen, allows the pressure to be maintained at normal indefinitely. Any increase in the amount of oxygen or the withdrawal of the nitrous oxid usually causes a sudden and marked elevation in blood-pressure, which persists for from five to fifteen minutes after the return to consciousness (Fig. 78).

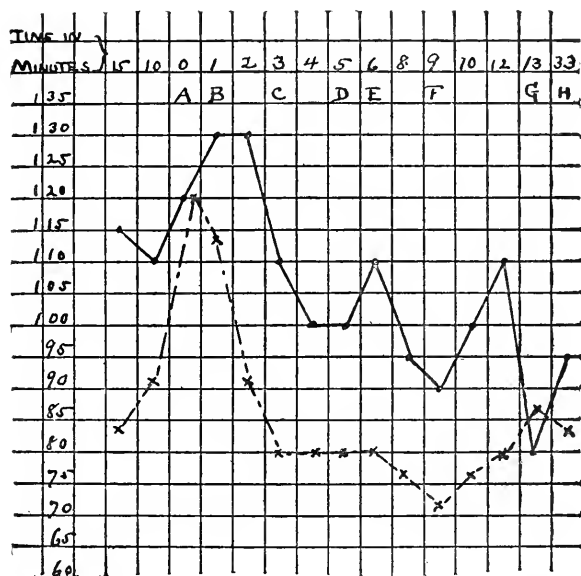


FIG. 80.—Male. Aged thirty-eight. Ethyl chlorid experimental anesthesia—duration twelve minutes. (A) Anesthesia begun. (B) Patient feels pain. (C) Still feels pain. (D) Another tube broken. (E) Complete anesthesia. (F) Anesthetic removed. (G) Consciousness returned. (H) Twenty-four minutes after removal of anesthetic, patient still has very marked reduced pain sense.

Ethyl Chlorid and Somnoform.—The administration of even 3 or 4 c.c. of these anesthetics has been followed by serious consequences; 5 c.c. has been known to produce death and any amount over this should be considered

dangerous. Its effect is that of powerful inhibition of both heart and blood-vessel tone, causing a progressive fall in blood-pressure. Ordinarily the pulse is greatly elevated and a dangerous hypotension is usually accompanied by a rapid and small pulse. The association of oxygen with ethyl chlorid tends somewhat to prevent the hypotension,

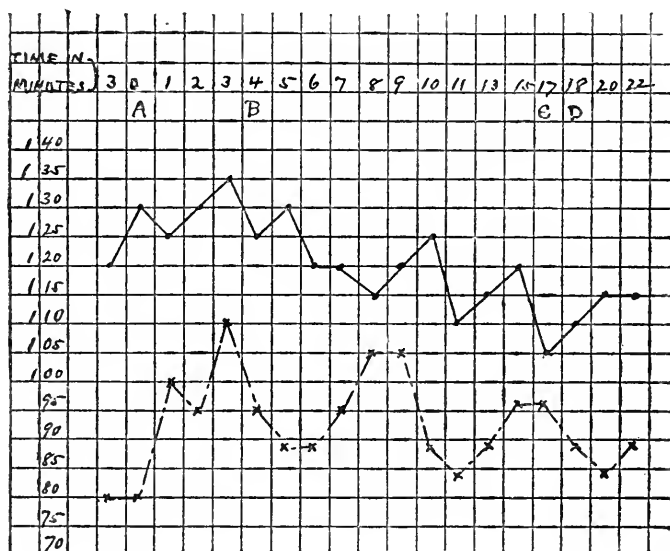


FIG. 81.—Somnoform analgesia. Duration seventeen minutes. Male. Aged twenty. Report by Dr. C. S. Tuttle. (A) Anesthetic begun. (B) Second tube broken. (C) Anesthetic terminated. (D) Oxygen inhalation begun. Note characteristic primary stimulation followed by subsequent depression of cardiovascular system as shown by gradual fall of systolic pressure from 135 mm. to 105 mm. in fourteen minutes.

thereby rendering the effect of this anesthetic less dangerous (Figs. 79, 80 and 81).

Spinal Anesthesia.—See Lumbar Puncture, page 358.

Administration of Salvarsan.—The administration of this remedy properly employed has but slight effect upon systolic pressure, although a short time afterward there

usually occurs a slight rise which may persist for a few days. The effect of efficient treatment of the infection, particularly in those showing aortic involvement, is followed by a gradual reduction in the systolic pressure. This would suggest the value of repeated blood-pressure observations in demonstrating the efficiency of specific treatment in certain cases.

Transfusion Operation.—The introduction of a volume of blood into the circulation may, if the transfer is accomplished with undue rapidity, cause a right-sided hypertension.¹ This condition is indicated by prostration, sweating, slight nausea, air hunger and great uneasiness. These symptoms can usually be controlled by less rapid transfusion or temporary cessation of the flow. There is no permanent alteration in pressure due to change in blood volume.

Relation of Blood Composition to Surgical Operations.—The data presented by Münzer² appears to demonstrate that many cases of anemia are distinguished by a lymphocytosis and a hypotension, and that these cases stand general anesthesia badly. He warns against prolonged operations under general anesthesia in such persons. W. J. Stone³ finds an increase in pulse pressure, in profound anemias, and cites three cases, in which the systolic pressure varied between 120 and 135, with pulse pressures between 45 and 65.

OPHTHALMOLOGIC DATA

For nearly ten years the value of sphygmomanometer readings in ophthalmology have been receiving increasing

¹ F. F. Simpson, *Jour. A. M. A.*, Sept. 11, 1915, lxx, 11, p. 941.

² *Medizin. Klinik.*, Dec. 14, 1914, lx, 50.

³ *Jour. A. M. A.*, Oct. 4, 1913, lxi, 14, p. 1256.

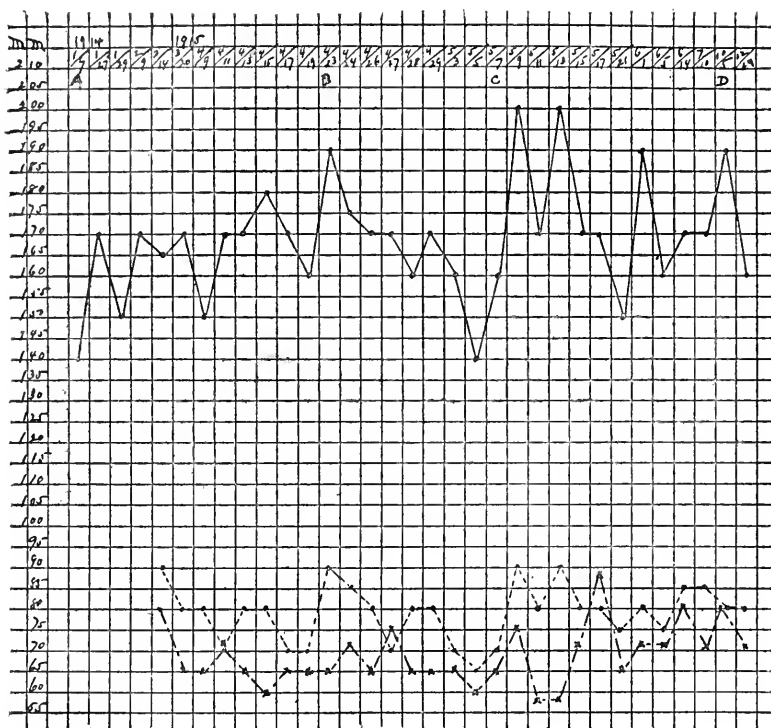


FIG. 82.—Age about seventy, female. Arteriosclerosis and chronic articular rheumatism. Acute glaucoma. (A) Patient reported having a dry cough for two weeks, which continues. Was physically weak, dyspneic and cyanosed. Examination shows dilated heart with weak muscle sounds, cyanosis, edema of legs and moderate pulmonary edema at both bases. Response to treatment very slow with several relapses. Patient progressed gradually but finally succeeded in getting around in the course of two months. (B) Patient developed acute glaucoma of right eye. Confined to bed. Treatment directed toward a reduction of systemic pressure by rest, sweats and purging with marked effect on systolic pressure, but very little upon the pulse pressure as shown in the chart, therefore this treatment, while benefiting the eye condition, did not actually relieve the cardiac overstrain. (C) General condition good. Up and about. (D) Has been taking digitalis in small doses irregularly for two months. Advised stopping digitalis and placing on routine treatment.

recognition and study, until now we know that there are conditions that are closely associated with high blood-pressure. These are retinal hemorrhage and glaucoma.

Glaucoma.—Fox and Batroff,¹ Dunn² and Jackson³ all dwell upon the importance of the relation of elevated blood-pressure to ocular glaucoma. Dunn states that he has never seen a case of essential glaucoma (Fig. 82) except in early youth, in which there has not been an elevated systolic pressure, and he quotes Frinke, who found pressures between 140 and 210 mm. Hg. in fourteen out of fifteen cases, and mentions a case of a reduction in pressure from 200 mm. Hg. to 175 and 178 resulted in a favorable change in an eye as shown by a reduction in intra-ocular tension from T.3 to T.1. It is safe to say that no case can be considered as thoroughly studied until the blood-pressure has been ascertained.

Neuroretinitis and Retinal Edema.—L. C. Peter⁴ studied the blood-pressure in 104 cases of chronic interstitial nephritis with high pressure, which showed either retinitis or neuroretinitis; and for purposes of comparison, nine cases of syphilitic origin, with an average systolic pressure of 132, and three cases of chronic parenchymatous nephritis with a systolic pressure of 132.

The average pressure in twenty of the 104 cases of retinitis was 165 mm. Hg., in fifty-nine cases of neuroretinitis it was 185 mm. Hg., in three cases of albuminuric retinitis, 190 mm. Hg., in six cases of hemorrhagic retinitis, 205 mm. Hg. and in three cases of papillitis, 225 mm. Hg. Thirty-

¹ 1908 ref.

² *Arch. Ophth.*

³ *Am. Jour. Ophth.*, December, 1909.

⁴ *Penna. Med. Jour.*, March, 1911.

three of the ninety-four kidney cases occurred in men and sixty-one in women. Peter concludes that arterial hypertension is the chief cause of these classes of ocular disease and that in them the use of the sphygmomanometer is clearly indicated. Its revelations are not only needful to a proper understanding of the pathologic conditions, and suggestive in prognosis and treatment, but they will also prevent blunders, which without this restraining influence, would be inevitable. According to Fox and Batroff the blood-pressure should be carefully and systematically studied in all eye cases past the period of middle life.

Retinal Hemorrhage and Systolic Pressure.—The studies of Fox and Batroff¹ were directed largely toward demonstrating the relation between retinal hemorrhages and high arterial pressure. From a study of 100 cases they concluded that “the true or exciting cause of these hemorrhages in a very large proportion of the cases is a sudden transient or persistent abnormal elevation of the arterial pressure.” And further that “the blood-pressure should be carefully and frequently studied in this class of ophthalmic cases; first, with a view to determining the presence of one of the most frequent causal conditions; second, to permit us to intelligently direct the treatment. The oculist, therefore, often being the first to be consulted, should study these patients with the internist, in order that the most comprehensive knowledge possible should be available for the sufferer.”

The summary of the findings of Fox and Batroff's series of 100 cases of hemorrhage is as follows:

Eighty per cent. occurred coincidentally with other dis-

¹ *Ophthal. Rec.*, October, 1908.

ease conditions in which high blood-pressure was the rule. The majority of retinal hemorrhages were found in persons suffering from chronic interstitial nephritis, 40 per cent.; the next most common relation was arteriosclerosis, 27 per cent.; and as is well known the eye-ground phenomena are often very early to appear in chronic interstitial nephritis and arteriosclerosis. Similar vascular changes, associated with high blood-pressure, may be observed at times before these diseases can be diagnosed by other clinical symptoms. High systolic pressure is also a cause for subconjunctival hemorrhage and is so closely associated with glaucoma that it should be regarded as an active factor in the development of this disease.

This author urges that, in order not only to prevent, but also to treat rationally the more serious eye conditions, routine blood-pressure studies should be made in all cases of intra-ocular disease of non-traumatic origin.

Peter's conclusions are as follows:

1. Arterial hypertension is the chief cause of the eye-ground phenomena observed in chronic interstitial nephritis and arteriosclerosis.

2. Similar vascular changes, associated with high blood-pressure, may be observed at times, before these diseases are diagnosed by other clinical symptoms.

3. It frequently acts as a cause for subconjunctival hemorrhage and is so closely associated with glaucoma that it should be regarded as an active factor in the development of the disease.

4. It probably will help to explain the phenomena of intra-ocular hemorrhage after cataract extraction.

5. In order to prevent and to treat rationally the more

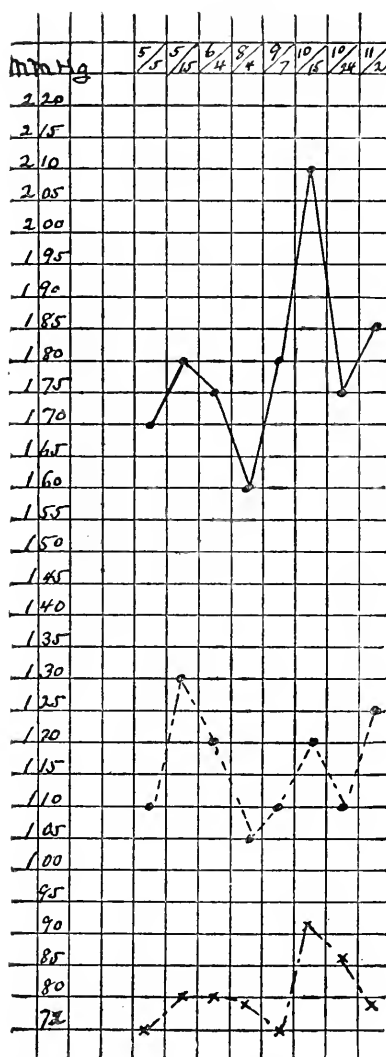


FIG. 83.—Male. Aged fifty-seven. Has never used alcohol or tobacco in any form. Life has been a hard one and there have lately been several severe mental shocks. Has long been a sufferer from a chronic cystitis. There is some myocarditis as shown by irregularity in force and volume of pulse. Ophthalmoscopic examination of eye-grounds shows nothing abnormal. The urinalysis shows evidence of contracted kidney and indicanuria is persistent. Patient's symptoms are chiefly gastric except for a series of ocular

serious eye conditions, routine blood-pressure studies should be made in all cases of intra-ocular disease not traumatic in origin.

Peter calls attention to another group of cases in which hypertension plays an important rôle, namely, spasm or ataxia of the retinal artery or branches, which was first brought out by Zentmayer in 1906 (Fig. 83).

In corneal ulcers the blood-pressure test may give us information as to why the treatment does not succeed, the explanation often being the presence of chronic kidney disease.

Surgical Aspect.—As a general rule, the higher the systolic pressure the less favorable is it for surgical procedures on the eye. A high blood-pressure will tell when not to operate in senile cataract, and when the danger of hemorrhage has been reduced by preliminary blood-pressure reducing measures. Such cases may have their surgical prognosis materially improved by preliminary medical treatment, directed toward amelioration of the excessive pressure. Blood-letting is most beneficial.

SHOCK¹

In spite of an immense volume of literature bearing on the problem of shock, and in spite of the many theories as

attacks which have been recurring with varying frequency for many years. The ocular attacks are described as follows: An effect likened unto crisscross illumination or waves of light, beginning at the left, in both eyes, and gradually advancing until one-half of each retina is so involved. This condition gradually fades and entirely disappears within ten minutes. During these attacks, if the patient views a direct light, there is marked tendency to the development of complimentary colors in the halo which appears upon the affected side of both retinae. There is no headache, dizziness or any other symptom. The condition is explained upon the ground of a persistent intestinal toxemia producing a temporary disturbance in blood-supply to the arteries of the retinae.

¹ Bibliography at end of chapter.

to its cause, and the mechanism of its production, it appears that the situation, as far at least as the ultimate solution of the problem is concerned, remains clouded.

Many theories have been advanced, since the work of Crile in 1903, each of which has both its exponents and opponents, owing chiefly to the inability of any one theory to meet all the conditions involved in the complex phenomenon known as surgical shock. So, at the present writing, it does not appear, from the data at hand, that the final solution of the problem has been reached.

Summarily speaking we recognize five principal theories as to the origin of shock.

1. Vasomotor exhaustion and paralysis (Crile, Mummery).
2. Cardiac spasm and eventual failures (Boise).
3. Inhibition of the functions of all the organs (Meltzer).
4. Acapnia or deficiency of CO_2 in the blood (Yandall Henderson).
5. Morphologic changes in the nervous system (Gray and Parsons).

That no one of these is adequate is shown; 1. By the researches of Seelig and Joseph and of Seelig and Lyon who show that the vasomotor center is not paralyzed in shock and that the arteries are contracted.

2. W. G. Porter and F. G. Mann have demonstrated that stimulation of a nerve trunk, even when prolonged, fails to cause its complete exhaustion.

3. Mann has also shown that degenerative changes in the central nervous system are the result and not the cause of shock.

4. That inhibition of the function of all organs has been

refuted by the resuscitation of shocked, exsanguinated animals by transfusion.

5. Wainwright and also Short have rendered the heart-spasm theory impossible by demonstrating the excellent recovery of the heart in shock by the operation of transfusion.

6. While Short demonstrates that acapnia is not usually present in surgical shock and therefore can only be depended on to explain a few cases.

Clinically shock may be divided into, first, anaphylactic shock, which is a laboratory phenomenon, more of academic than of practical importance. Second, toxic shock which differs in no way from surgical shock, except etiologically, with which its symptoms and disturbances are identical. Third, surgical shock. This condition irrespective of its etiology and the mechanism of its production, is undoubtedly vasomotor, an essential feature of which is a radical disturbance in blood-pressure, probably due to an interference in the normal distribution in the blood. Janeway and Jackson have shown experimentally that this disturbance is of such a character that the normal quota of blood upon the arterial side of the circulation is diminished, and that this diminution is maintained so that, as a consequence, even after the original cause of the disturbed distribution of the blood, whether of a mechanical, toxic or inhibitory nature, is removed, the abnormal distribution of the blood upon the arterial side of the circulation may not only persist but, in fatal cases, continues until death.

Much experimental work tends to sustain the theory that the low pressure found in shock is due to the retention of blood within the vessels on the venous side of the

circulation, particularly of the splanchnic areas, whereby the volume of blood in the arterial tree is reduced to such a degree that it mechanically interferes with the normal systole which even the contracted arterioles and small arteries, that have been demonstrated to exist during shock, fail to relieve.

C. F. Mann explains the loss of circulatory fluid in a different way, although the end-results are the same; namely, a reduced systolic and a diminished pulse pressure.

He believes "that the clinical signs of shock which appear after section of the abdomen and exposure of the viscera are due to a loss of circulatory fluid. This loss of fluid is not dependent on any primary impairment of the medullary center and takes place at a point beyond the control of the vasomotor mechanism. The causes for this loss of fluid are apparently the same as those which determine the accumulation of fluid in any other irritated area and produce the signs of inflammation. The nervous system probably plays no greater part in the former case than in the latter."

This diminution of the volume of the circulating blood offers an explanation of most of the acknowledged facts. It solves the crucial problem of a low blood-pressure with constricted arteries, vigorous heart muscle and an active vasomotor center. The changes in the nerve cells are due to the failure of the proper blood-supply and the mental apathy is the result of the same. The small pulse and pale skin are just what we should expect in such a condition. The undoubted value of saline infusion into a vein or injection into the rectum also receives explanation.

H. P. Fairlie has reported some practical conclusions

drawn from a study of the reaction of cases in shock under chloroform and under ether.

The fall in pressure in shock under ether is as great as the fall under chloroform, while the response is slower. In severe cases of shock at the end of the operation, the blood-pressure shows a better response under chloroform than it does under ether.

After weighing all considerations Fairlie suggests that in the severe cases chloroform is the anesthetic of choice, but that the anesthesia should be first induced by ether and then carried on by chloroform. The value of these conclusions must stand alone, as I have been unable to find any other references on either side of the question.

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CHAPTER XXI

THE BLOOD-PRESSURE TEST IN OBSTETRICS

The universal employment of the sphygmomanometer and the routine study of normal and pathologic cases of pregnancy, during the past decade, have established a number of working rules which now serve as guides in diagnosis and prognosis, so that the sphygmomanometer now ranks with urinalysis in the examination of the pregnant.

In the blood-pressure test we have a most valuable means of detecting early toxemias, which often lead to the eclamptic state. Abnormal blood-pressure readings are one of the earliest indications of a departure from normal metabolism in the pregnant. This change may be evident before the development of any physical signs, or of any noticeable change in the urine. From a pathologic standpoint, it is evident that the close relation between the kidney and blood-pressure should be a valuable guide during this condition, since alterations in metabolism, the retention of waste products and the development of special toxins in the blood usually show themselves in a gradually rising blood-pressure, the one exception to this rule being those toxemias of hepatic origin in which the blood-pressure usually is not elevated (see page 392). These cases are, however, so few that they do not detract from the value and importance of the blood-pressure examination.

Routine blood-pressure observations should be made a part of the periodic examination during pregnancy.

The intervals between the tests should be shortened as the pregnancy advances, and, should abnormal symptoms appear at more frequent intervals. The test should not be omitted during the puerperium, as at this time the patient may develop serious toxemia and eclamptic attacks.

Pregnant patients should have the blood-pressure test applied at least as frequently as the urine is examined. It would be advisable to apply the sphygmomanometer as often as practicable. So employed, and with the records properly charted, blood-pressure tests may furnish a far more adequate indication of the seriousness of a pregnancy nephritis and the urgency of inducing labor, than the usual urinalysis.

THE NORMAL BLOOD-PRESSURE DURING PREGNANCY

Most observers agree that the average systolic blood-pressure during most of the period of pregnancy is in the neighborhood of 120. John C. Hirst¹ in the study of 100 cases obtained a general average systolic pressure of 118.

H. C. Bailey² arrived at the same average. While these same authorities look upon a persistent pressure of over 145 with suspicion, Arthur J. Benedict³ believes that a pressure of over 125 in pregnancy is abnormal and indicates toxemia.

I have private records of 205 observations made upon thirty-four cases of normal pregnancy, in which the diastolic and pulse pressure readings as well as the systolic were made simultaneously, with complete urinalyses. The following is a summary of the results obtained:

¹ *N. Y. Med. Jour.*, June 11, 1910, p. 204.

² *Surg. Gyn. and Obstet.*, Vol. xiii, 5, 1911, p. 485.

³ *Brit. Med. Jour.*, Dec. 3, 1910.

	Normal
Number of cases.....	34
Number of observations.....	205
Average systolic.....	117.5 mm. Hg.
Highest systolic.....	140.0 mm. Hg. (4 cases)
Lowest systolic.....	90.0 mm. Hg. (1 case)
Average pulse pressure.....	38.5 mm. Hg.
Highest pulse pressure.....	55.0 mm. Hg. (1 case)
Lowest pulse pressure.....	25.0 mm. Hg. (2 cases)
Average diastolic.....	79.0 mm. Hg.
Highest diastolic.....	95.0 mm. Hg. (1 case)
Lowest diastolic.....	65.0 mm. Hg. (2 cases)
Average pulse rate.....	85.1
Highest pulse rate.....	140.0 (1 case)
Lowest pulse rate.....	72.0 (8 cases)

The following is a summary of 124 urinalyses made upon the same group:

Thirty-four normal cases.

One hundred and twenty-four examinations.

Eight or 23.5 per cent. (of cases) showed no albumin at any time.

Sixteen or 47.9 per cent. showed albumin during first five months.

Sixteen or 47.9 per cent. showed albumin during last four months.

The albuminuria bore no definite relation to the pressure reading or the period of gestation.

The specific gravity ran from 1005 to 1036—average 1018.

Twenty-one or 61.7 per cent. showed at some time casts or cylindroids in small numbers. Three showed granular casts which had apparently no significance. Four cases which showed indican had an average systolic pressure of 105 mm.

The opinion is generally held that there is but slight change in the systolic pressure until the later months of pregnancy and that then a slight rise occurs.

In four abnormal cases under my care involving six pregnancies, the average systolic pressure was 158, while the urine ranged from a mere trace to a solid mass of albumin in the tube after boiling. The microscope showed all varieties of abnormal morphologic elements.

Naturally the individual readings must vary greatly within certain limits, as these patients are subject to the

same influences which affect the reading in other individuals, so that rapid variations are probably insignificant unless they exceed 30 mm. above the average and remain persistently at or above the high normal limit.

EFFECT OF LABOR ON BLOOD-PRESSURE

During the first stage of labor, at the recurrence of pains there is a marked rise in pressure which falls in the intervals, but usually during the first and second stages the level remains at from 140 to 150 even between pains (Bailey).

Hirst had noted that a fall of pressure coincides with rupture of the membranes, sometimes amounting to 50 or more millimeters, usually accompanied by marked relief from headache and epigastric symptoms. This is temporary, as the pressure gradually rises as labor continues. There is a second fall of 60 to 90 mm. immediately after childbirth, which is also temporary, the pressure soon returning almost to the level attained before birth. Profuse hemorrhage or the supervention of exhaustion will interfere with this rise, the degree of reduction in pressure indicating the seriousness of these complications. Obstetric operations, according to Cook and Briggs,¹ which involve the introduction of the hand into the vagina or uterus, and instrumental deliveries, cause a sharp reflex rise which has been known to result in rupture of a cerebral vessel.

COMPLICATIONS AFFECTING BLOOD-PRESSURE DURING PREGNANCY

Albuminuria.—Albumin in small quantities appearing in the urine during pregnancy cannot be considered pathologic, as in a large percentage of cases it appears from time

¹ *Johns Hopkins Hosp. Rep.*, 1903, Vol. xi, 451.

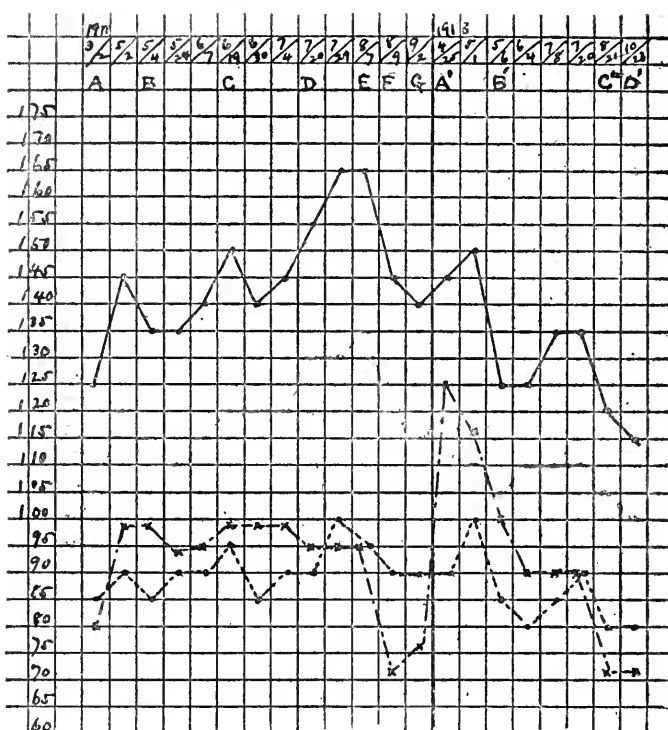


FIG. 84.—Aged twenty-five. Two pregnancies: the first toxic, the second not.

At (A) five months pregnant (March, 1911). Normal to this date.

At (B) beginning development of edema, headaches, albuminuria unrestrained by dietetic regulations between (B) and (C).

At (C) hot-packs begun, with slight reduction in systolic pressure but failure to relieve cardiac over-action as shown by the continued elevation of pulse pressure.

(D) Gradual failure of eliminative measures. Symptoms more marked but not considered urgent.

(E) Labor induced on account of continued elevation and rapidly increasing nervous symptoms, high-grade albuminuria and kidney failure.

(F) Delivered without serious complications.

(G) Final examination. Patient passed from observation still showing edema, dyspnea, with albumin and casts in the urine.

(A¹) Between five and six months pregnant (April, 1913) with marked signs of kidney failure. Dietetic and eliminative measures instituted.

to time and seems of itself to have no relativity to the subsequent course of the case.¹ In a series of thirty-four normal cases which I have recorded, albumin appeared in sixty-two examinations of 50 per cent. The combination of traces of albumin with a rising systolic pressure will give the obstetrician pause, and a *rising pressure plus albumin* which fails to respond to treatment is an indication for the forced termination of pregnancy² (Fig. 84).

Pernicious Vomiting.—Donaldson's³ observations lead to the belief that, while this condition may demand the induction of labor, the blood-pressure is not elevated; which plainly suggests that the toxemia in these cases differs from that occurring in eclamptics.

Glycosuria.—Donaldson reports nothing abnormal in the blood-pressure readings in glycosuria, which coincides with my own experience and is shown in the accompanying chart (Fig. 85).

Hemorrhage.—Wallich⁴ considers that elevation of pressure during pregnancy tends to hemorrhage and that this accounts for the hemorrhagic lesions so common in eclampsia. On the other hand, estimation of blood pressure is important in suspected hemorrhage as in cases of ectopic

(B¹) All symptoms subsided. Patient on rigid diet and hygiene. Condition satisfactory. Urine shows only slight evidence of renal irritation.

(C¹) Delivered at term.

(D¹) Two months after delivery. Condition excellent. Urine practically normal, no evidence of kidney insufficiency.

Note, January, 1916, patient has not been pregnant again, has enjoyed good health and shows no evidence of kidney involvement.

¹ Franklin S. Newall, *Jour. A. M. A.*, Jan. 30, 1915, lxiv, 5.

² M. Donaldson, *Jour. Obstet. and Gyn.*, London, September, 1913, xxiv, 3; and J. D. Lippincott, *Jour. A. M. A.*, Aug. 10, 1912, lix, 6.

³ *Loc. cit.*

⁴ V. Wallich, *Annal. de Gynae. et d'Obstet.*, November, 1913, xxxix, No. 11.

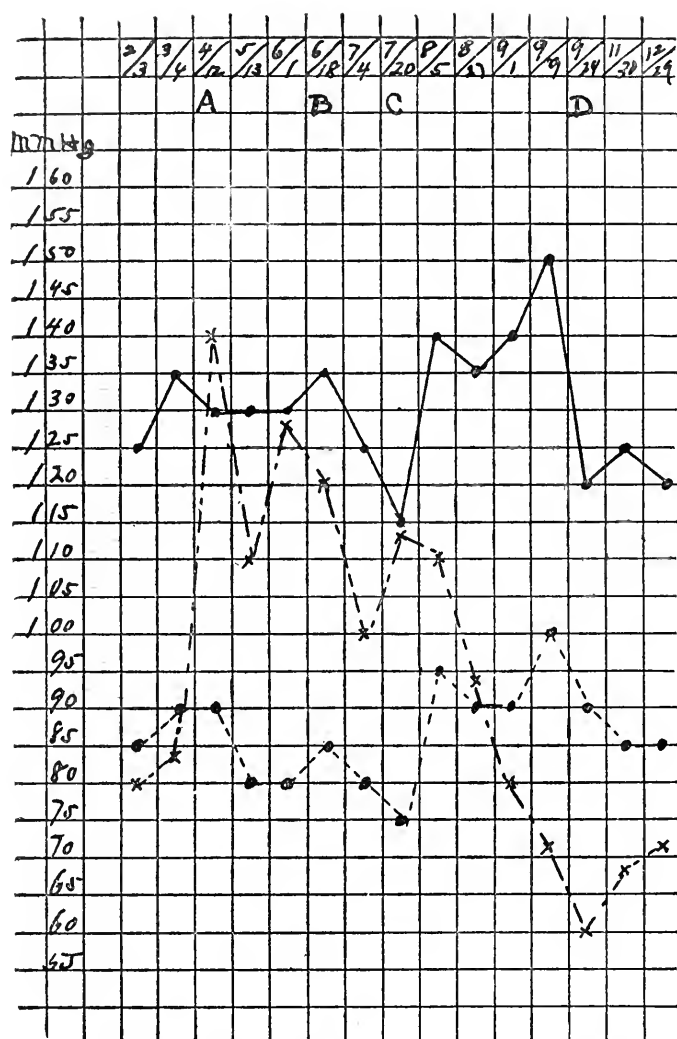


FIG. 85.—Female. Aged thirty-two. Primipara. Glycosuria. Albumin appearing the third month (A) and sugar was first noted the fifth month (B) and reached $1\frac{1}{2}$ per cent. Dietetic measures were followed by its disappearance at (C) after which it did not return. Patient was delivered Sept. 24 (D) of a normal baby and had an uneventful convalescence. Important points in this case appeared to be a rather marked depression of systolic pressure during the period of glycosuria, its rapid disappearance and continued absence up to and after delivery. In spite of the rise in systolic pressure to 150 this patient had at no time showed signs of kidney insufficiency or toxemia.

gestation and other surgical complications, as a sudden and continued drop accompanied by a rising pulse rate is significant of persistent hemorrhage. If, after noting these significant changes, they are, by subsequent observation, found to be arrested or to have become reversed, it is safe to infer that the hemorrhage has ceased. P. Ballard¹ in discussing the significance of pressure changes to hemorrhage states that the systolic pressure in hemorrhage may vary within a wide range, but that a diastolic pressure of 50 mm. appears to be the lowest point at which the heart is able to keep the blood circulating.

Low Pressure at Term.—Franklin S. Newall² considers that an abnormally low pressure at term (100 or less) in the absence of toxemia, suggests that the patient is in poor condition and is likely to react unduly to the strain of labor, so that under these conditions every effort should be made to avoid excessive strain, suffering and shock.

Toxemia and Eclampsia.—John Cooke Hirst³ states that the earliest and most constant sign of toxemia in the latter half of pregnancy is a high and constantly rising blood-pressure (Fig. 86), and this symptom *precedes* albuminuria and all the constitutional signs of an impending eclamptic attack.

This most serious and often fatal complication of pregnancy and the puerperium is recognized as the most fruitful cause of elevated systolic pressure, although a high blood-pressure is not the invariable accompaniment of this condition. J. C. Hirst states:⁴ “cases are seen undoubtedly

¹ *Arch. Mens. d'Obstet. et de Gynae.*, December, 1914, iii, 12.

² *Jour. A. M. A.*, Jan. 30, 1915, lxiv, 5, p. 393.

³ *N. Y. Med. Jour.*, June 11, 1910.

⁴ *Penna. Med. Jour.*, xviii, 8, 1915, p. 650.

toxic, even in eclampsia, with a pressure of 130 or lower." These are probably of liver origin, and are usually more serious than those of nephritic origin. The blood-pressure test may serve as a basis for differentiation.

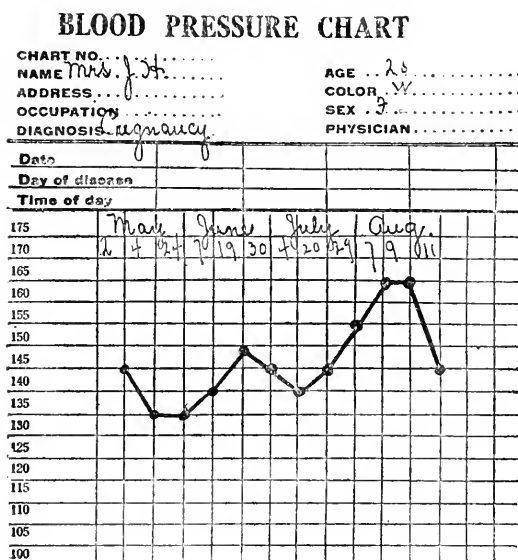


FIG. 86.—May 2, case showed albumin, scanty urine, headaches and dizziness, symptoms relieved by hot pack and purgation at irregularly repeated intervals. Premature induction of labor advised but declined. Normal delivery on August 9. Treatment controlled subjective symptoms, but did not much affect the tendency to a rising blood-pressure.

The prevailing opinion is that eclampsia is usually accompanied by a pressure of over 200 mm. Hg.¹

Two cases have been reported by Hirst with pressures of 400 to 420 and, surprising to record, they both got well, so that extremely high pressures are not necessarily fatal,

¹ J. O. Evans, *N. W. Medicine*, June, 1912, iv, 6, p. 183, and H. Vaquez, *La Semaine Medicale*, 1907, ii, p. 121, and Bailey, *loc. cit.*

although clinically the lower the pressure in actual eclampsia, the more favorable the prognosis.

If abnormally high pressure persists in the third stage, or there is little or none of the normal decline, measures for relief must be instituted almost as urgently as if the seizures were present.

T. M. Green¹ conveniently divides toxemia of pregnancy into three divisions:

First, moderate increase in blood-pressure.

Second, marked increase in blood-pressure.

Third, extreme increase of blood-pressure.

To these may be added the fourth, which is suggested by the studies of Hirst and of Baily, namely: extreme eclamptic condition in which the blood-pressure may be low.

In the first two, symptoms disappear and blood-pressure falls after delivery. In the third and fourth, blood-pressure continues abnormal, and the disease usually progresses to a rapidly fatal termination.

To summarize our present knowledge of the relation of blood-pressure findings, it is believed that:

First, the normal blood-pressure in healthy pregnant women will average close to 118 mm. A slight increase over these figures but below 150 mm., is to be expected in the last month of pregnancy.

Second, blood-pressure in toxemia in the first half of pregnancy, associated with pernicious vomiting, is usually low.

Third, blood-pressure in the latter half of pregnancy, associated with albuminuria and eclampsia, is invariably high.

¹ *Boston Med. and Surg. Jour.*, Apr. 28, 1910.

Fourth, a high and rising blood-pressure is an invariable and very often the earliest sign of toxemia in the latter half of pregnancy.

Fifth, the actual height of the pressure is not of itself necessarily an indication of the gravity of the prognosis.

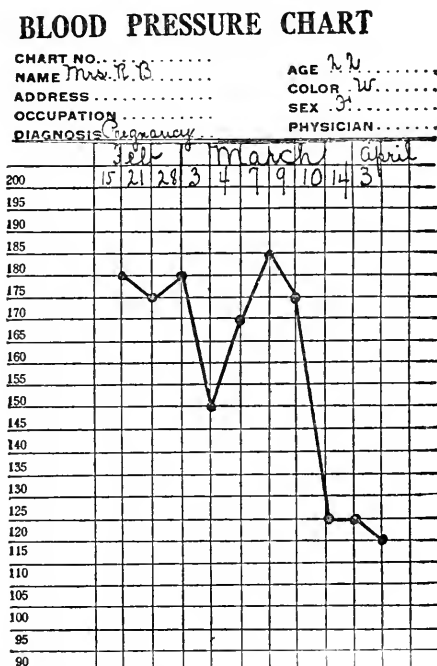


FIG. 87.—Term calculated to March 30, labor induced March 9. February 5, ankles edematous, marked gastric irritation, large amount of albumin in urine. This condition not relieved by treatment. March 7, urine boiled nearly solid. March 9, labor induced; March 10, delivered; April 3, albumin absent, patient normal.

Sixth, cases of eclampsia which recover, usually show a rapid fall of pressure after delivery, while the high-pressure cases which eventually die have a persistently high pressure.

Seventh, usually in eclampsia, the pressure remains high

for forty-eight hours after the birth, then begins to subside and reaches the normal of from 118 to 124 mm. in from seven to ten days after delivery.

Eighth, upon the rupture of the membranes, in toxic cases, there is an immediate fall of pressure of from 60 to 90 mm. This fall is temporary only, but is attended with marked relief in the headache and epigastric pain these patients so frequently complain of. However, after some hours the pressure returns toward its original level.

Ninth, there is a second fall of from 60 to 90 mm. after the child is born. This again is only temporary, as in from fifteen to thirty minutes, if a patient has not bled profusely, the pressure returns to about its level before birth.

Tenth, as far as it is possible to lay down any rules in these cases we may say that a blood-pressure of below 125 mm. could be disregarded, a pressure of from 125 to 150 mm. needs careful watching and moderate eliminative treatment, and that a pressure of over 150 mm. needs usually active eliminative treatment, and will in all probability, especially if it shows a tendency to climb higher, require the induction of premature labor (Fig. 87).

EFFECTS OF DRUGS UPON BLOOD-PRESSURE DURING PREGNANCY AND LABOR

Ergot has apparently no effect on either normal or abnormal pressures.

Pituitrin produces a rapid temporary elevation of from 25 to 40 mm. and occasionally a violent rise in pressure, there are exceptions where it has no effect. The action is most rapid when administered intravenously. This sudden elevation may be harmful in toxemic cases and those with

cardiorenal complications. The elevation persists for from thirty to forty minutes and is often followed by a sudden fall to below normal.¹

Scopolamin and Morphin Narcosis.—Joseph L. Baer² reports an average fall of 5 mm. in a clinical study of sixty cases.

Chloroform.—This anesthetic is considered dangerous by Whipple,³ both because of its blood-pressure-reducing propensity and because of the frequency of subsequent liver necrosis following its use, which would tend to favor toxemia.

¹ S. S. Arbuck and A. J. Rongy, *N. Y. Med. Jour.*, May 2, 1914, p. 878.

² *Jour. A. M. A.*, lxiv, 21, May 22, 1915, p. 1723.

³ G. H. Whipple, *Jour. Exp. Med.*, Mar. 1, 1912.

CHAPTER XXII

BLOOD-PRESSURE ESTIMATIONS IN PHYSICAL EXAMINATIONS FOR LIFE INSURANCE

Prior to 1907, when the publication of Janeway's book on blood-pressure gave a new impetus to blood-pressure studies, little or no use was made of the sphygmomanometer in the examination of applicants for life insurance. In the following year (1908) the present author undertook a statistical study of this subject with a view to determining the attitude of life insurance companies toward the blood-pressure test. A circular letter containing a series of questions was addressed to forty-six medical directors, and a summary of these replies embodied in a paper read before the American Association of Life Insurance Examiners at their Atlantic City session in June, 1909.¹ While pursuing this investigation, I had an opportunity of addressing the Philadelphia Medical Examiners' Association on March 2, 1909, upon this same subject² at which time I said, "Recent increase in clinical data, which is the result of finer methods of observation (of blood-pressure), demand greater recognition by the practicing physician and the medical examiner" and further, "the sphygmomanometer is a valuable aid in determining arterial tension, and in differentiating diseases of the cardiovascular system."

The replies of thirty-two medical directors, to this circular letter, developed the interesting fact that at that

¹ *N. Y. Med. Jour.*, July 23, 1910.

² *N. Y. Med. Jour.*, May 22, 1909.

time no insurance company of any note employed the blood-pressure test in all examinations, although 67 per cent. made partial use of it and found it valuable in special conditions where impairment was suspected, either because of information previously acquired, or in overweights, in those of special occupations, and in those in which the result of part of the examination suggested the presence of an elevated systolic pressure in connection with cardiac, vascular or kidney lesions. The article concluded with the following: "Bearing in mind that $25\frac{1}{10}$ per cent. of insured risks in one company—were due directly or indirectly to a pathologic increase in blood-pressure, I submit . . . , in the sphygmomanometer we have a ready and accurate means of determining a greater number of such cases, than is possible by the means heretofore at the disposal of the examiner for life insurance.

"This alone, should be sufficient argument for the universal adoption of this test, which would result in much financial saving to the life insurance companies."

Following this lead J. W. Fisher, in 1911,¹ after employing the test for a couple of years stated that: "No practitioner of medicine should be without a sphygmomanometer. He has in this instrument a most valuable aid to diagnosis. The sphygmomanometer is indispensable in life insurance examinations, and the time is not far distant when all progressive life insurance companies will require its use in all examinations of applicants for life insurance," practically a repetition of the author's prediction of two years before.

¹ Quotation taken from article by same author, *Jour. A. M. A.*, Nov. 14, 1914, lxiii, No. 20.

A general application of this fact has been so rapid that to-day, with possibly one or two exceptions, the blood-pressure test is employed by all American life insurance companies and by the majority of those abroad. Indeed the medical directors are now agitating in favor of the diastolic determination as well (see Chapter V, page 89, for importance of diastolic blood-pressure tests).

A recent and able argument in the same cause, by J. S. Lankford, appears in the May 23, 1914, issue of the *Medical Record* (Vol. 85, No. 21).

VALUE OF THE TEST IN LIFE INSURANCE

By this test we may very early detect signs of beginning pathologic change in the cardiovascular system and in the kidneys, often before there is any demonstrable evidence of departure from normal either in the physical signs, personal history or urine. This is chiefly because the apparent character of the pulse, and the examination of the superficial vessels, does not always portray the actual condition of the general arterial tree or the degree of arterial tension.

Clinicians are agreed that tactile estimation of blood-pressure is most unsatisfactory, and in many cases unreliable and often misleading. Even the most experienced have been unconsciously led into grave error by depending upon tactile sensations when the sphygmomanometer could have been employed.

To quote from Wm. Russell we find the following very significant statement: "I must, however, again add a warning note to the effect that feeling the radial pulse is not always a reliable guide as to what the blood-pressure will read. I have two such cases under observation, the

radial being neither hard nor incompressible, and yet in both there is a steady reading of over 200 mm. Hg."

Many times we may feel a soft and compressible radial where there exists marked sclerosis of the aorta and of the splanchnic area. Here the blood-pressure test alone reveals the true situation. In other instances the reading of the sphygmomanometer may explain the significance of an apparently simple headache, a mild attack of indigestion, or transitory attacks of vertigo in an apparently healthy individual, by demonstrating that these cases have suffered from a long-continued toxemia, which has resulted in an unsuspected pathologic change in the cerebral or general arterial system.

From the clinical standpoint, it is now well recognized that such pathologic changes may be present in the cardiovascular and renal systems, long before any subjective symptoms are complained of by the individual, or if any complaint is made, the symptoms are usually attributed to some trivial cause.

John B. McAlister¹ states that insurance statistics of 1247 cases of all ages, in which the pressure was 150 mm. or over showed a mortality of two and one-half times that of the general mortality for the same period. Of these, 891 or more than three-fifths, had no impairment recorded other than the high pressure. The important bearing of this upon the character of insurance risks is shown by Thayer and Brush, who after a critical study of 3894 persons at Johns Hopkins Hospital draw the following conclusion: "it seems to us that there can be little doubt that the main etiologic factor in the development of hyperplastic thickening of the

¹ Med. Council, July, 1914.

intima, which constitutes so important an element in arteriosclerosis, is overstrain of the vascular wall from continued or intermittent high tension, whatever the ultimate cause may be."

Formula to Estimate Normal Pressure.—To furnish a ready method of determining the average normal blood-pressure at a given age, the author has suggested a formula, based upon a large number of observations of his own and of others, which can be universally applied. The average obtained by the formula agrees closely with the experience of most observers, and since its first publication in 1910,¹ it has been extensively quoted and is now employed by at least one insurance company (The Provident Life and Trust Company, Philadelphia). As originally suggested, it was as follows: "Consider the average normal systolic blood-pressure in the male at age twenty to be 120 mm. of Hg.; for each year of life thereafter $\frac{1}{2}$ mm. to 120." Later it seemed advisable to eliminate the fraction, and this was done by changing the phraseology to read as follows: "Consider the normal average systolic blood-pressure of a male, age twenty to be 120 mm., then add 1 mm. to every additional two years of life." In both the formulas the result is the same, thus at the age thirty the normal average systolic blood-pressure would be 125, at sixty, 140 mm., etc. It is sufficiently established to pass without question that the normal average blood-pressure for females at the same ages is approximately 10 mm. less than for the male.

Permissible Variations.—It is not sufficient to establish a normal average with which to rate the risk but it is

¹ "The Sphygmomanometer and its Practical Application," Pilling Co., Philadelphia, 1910.

necessary also to determine what variations above and below this shall be accepted as normal. Unfortunately, with the evidence at hand this question cannot be definitely answered, for existing statistics do not agree.

Janeway regards with suspicion any systolic pressure which remains, before middle life, persistently above 145 mm. and places the maximum safe level at 160 in later years.

In 90 per cent. of a series of 656 healthy young men, Barach and Marks¹ found that the systolic blood-pressure was 150 mm. Hg., and in 96 per cent. of the 338 persons, the diastolic pressure, read at the fifth point, did not exceed 100 mm. Hg., while in 87 per cent. of a series of 312, the diastolic pressure, read at the fourth point, did not exceed 100 mm. Hg. In 88 per cent. of a series of 629, the pulse pressure ranged between 20 and 70 mm. Hg.

J. W. Fisher² is firmly of the opinion that a systolic pressure that is persistently above the average (see page 114) by more than 15 mm. for a given age, "should excite suspicion and call for further examination, as we find that an abnormally high pressure has been noted in about 6.5 per cent. of all applicants declined for insurance in the Northwestern Mutual Life Insurance Company."

As far as can be gathered from many published reports of blood-pressure tests, a maximum diurnal variation of 36 mm. in normal individuals does not exceed normal. If we accept this, then a variation of 17 mm. above or below the normal estimated average may be allowed. Thus at age twenty any reading of over 137 or below 103 would call for explanation, while at age fifty, the permissible variation

¹ H. H. Barach and W. L. Marks, *Arch. Int. Med.*, Apr. 15, 1914, xiii, 4.

² *Jour. A. M. A.*, 1914, No. 14.

lies between 152 mm. and 118 mm. In all determinations of blood-pressure, the factor of the diameter of the cuff employed must be considered, assuming of course that the accuracy of the instrument itself is beyond dispute.

At the present time the accepted standard for the width of cuff is between $4\frac{1}{4}$ and 5 in. (11 cm. to 13 cm.). A cuff of narrower width gives higher readings proportionately to the narrowness of the cuff, and the height of the pressure.

Application.—As a routine measure, the left arm should be employed and be bared to permit application of the cuff. Both patient and operator should be in comfortable positions, by preference the sitting posture. Time also should be allowed for the circulation to become quieted, as after rapid walking, stair climbing, etc.

Physiologic variations need not confuse the examiner, as they all occur within a range sufficiently restricted to prevent obscuration of the issue.

One should never fail to discount the psychic element which may be great. Fear and apprehension on the part of the applicant may occasion a sharp rise, so that in cases bordering on high it is well to disregard the first reading, as the second and third readings would likely be more normal.

Relation of Systolic, Diastolic and Pulse Pressures.—Generally speaking the systolic and diastolic and pulse pressures should approximate the relationship of 3:2:1, *i.e.*, S.P. 130; D.P. 86; P.P. 44; thus the pulse pressure is one-half of the diastolic pressure and one-third of the systolic pressure. To put it still more simply, any condition in which the pulse pressure multiplied by three greatly exceeds the systolic pressure is an indication of cardiac

overwork and suggests the probability of arterial and myocardial involvement. On the other hand should three times the pulse pressure be much less than the systolic pressure there is evidence of great physical or circulatory weakness. Persons who show these variations should be regarded with suspicion, even when the systolic pressure alone is approximately normal for the given age.

In chronic nephritis during cardiac compensation the pulse pressure is often very large, at times numerically approximating the diastolic pressure, while in aortic insufficiency very large pulse pressures are met; this variation is said to be pathognomonic of this condition. Thus in a case recently under observation the systolic pressure averaged 145, the diastolic pressure 40 (auscultatory, fourth phase) and the pulse pressure 105 mm.

Chronic Albuminuria and Blood-pressure.—In cases showing chronic albuminuria great importance is attached to the systolic blood-pressure, for, if after about ten years of recurrent albuminuria, the pressure is not found to be unduly elevated, the urinary findings may be considered far less grave than they would be otherwise. In this connection a case is now under my observation that was rejected for insurance over eighteen years ago, and yet the systolic pressure never has been observed to be higher than 130 and, although the urine still shows traces of albumin, he appears to be in good health. While this may be considered an extreme example, nevertheless it is to the point, as similar cases of shorter duration can be cited in large numbers. It is the experience of all who daily employ the blood-pressure test to encounter not

only albumin, but also casts, in persons with normal blood-pressure, and where the urinary findings are probably purely metabolic.

Nephritis.—Bearing in mind the difficulty of early diagnosis in cases of chronic nephritis by a single urinalysis, particularly in individuals apparently in normal health, the importance of a blood-pressure test will be apparent, because it is recognized that we cannot have permanent kidney change without a constant elevation in blood-pressure, and even in the presence of albumin or casts we may question their true significance. Here a persistently high blood-pressure, say 150 mm. or over, in an individual below middle age will settle the question at least in regard to the risk. The presence alone of scanty albumin and casts in the urine is not conclusive evidence of a diseased kidney, as these elements may come from any number of transitory and comparatively unimportant complications. The blood-pressure test will serve as a check, so that the applicant with a normal blood-pressure whose urine has occasionally shown albumin and casts will not immediately be rejected, and such individuals will get the benefit of the doubt and the company thereby be prevented from committing perhaps a grave injustice.

Overweights.—The overweights demand careful consideration by the insurance examiner. This is a group which shows an unfavorable mortality in life insurance statistics, particularly in the higher ages. It should be remembered that the amount of adipose tissue covering the vessels does not materially affect the reading, as cases of very large arms present readings of normal or even below, so that findings of high pressure should be attributed

to some other cause. In a person of moderate overweight in whom nothing in the physical examination or history indicates rejection, the final decision is often made upon the relation of the blood-pressure test. The risk is accepted when pressure is found normal and declined when the pressure approaches or passes the high normal limit.

Chronic Myocarditis.—This is probably one of the most difficult conditions to diagnose that is encountered in the course of insurance work. Its possible presence must always be borne in mind and every effort made to eliminate it in the examination, particularly in those past middle life, and in those presenting past history of hard physical labor, excessive brain work, alcoholism or syphilis. This will of course not be difficult to recognize when the disease has progressed sufficiently to affect the general health of the individual. It is in the early stages, where the usual methods of examination fail to reveal it, that the sphygmomanometer is of greatest value. In the early cases the systolic pressure need not be materially affected, so that recourse must be had to the functional tests of Graüpnér and Shapiro, and to a study of the diastolic and pulse pressures, by which changes in normal reserve of the heart and the strength and volume of its output can be estimated (see page 331).

Incipient Tuberculosis.—The presence of a slightly lowered blood-pressure accompanied by slight elevation in pulse rate, with or without fever, combined with a history of slight loss of weight, is very suggestive evidence of an existing pulmonary lesion. In tuberculosis the blood-pressure is usually low and the pulse pressure diminished.

In this connection Haven Emerson¹ states that hypo-

¹ *Arch. Int. Med.*, 1910.

tension is found in almost all cases of moderately advanced tuberculosis and that it has been found by many observers in early doubtful or suspected cases with or without physical signs of the disease of the lungs, and that it is considered by competent clinicians as a most useful sign. Cook also states that low blood-pressure, if persistently found in individuals or in families should put us on our guard for tuberculosis. In applicants of light weight and a blood-pressure of 100 or under and of poor family history, the risk is bad (see also page 203).

Blood-pressure in Relation to Mortality.—Dr. J. W. Fisher of the Northwestern Mutual Life Insurance Company,^{1, 2} has produced some very valuable work by drawing conclusions from a study and analysis of the mortality statistics of that company beginning in 1907 and continuing until August 1, 1913. This report amply confirms present opinions regarding the value of the blood-pressure test in detecting beginning disease of the cardiovascular and renal systems, and their influence on expected mortality.

From a study of 2661 insured taken from the actuary's tables giving blood-pressure readings between 140 and 149 mm. Hg., with 81.85 expected deaths, thirty-one actual deaths, a percentage of 37.87, which was slightly below the normal death rate of the company on exposure of two years. He shows another table of mortality records of 527 insured persons with a blood-pressure reading of 150 mm. Hg. and over, with 22.19 expected deaths and actual deaths twelve, which is about 35 per cent. in excess of the general average

¹ *Med. Rec.*, Oct. 21, 1911.

² *Jour. A. M. A.*, Nov. 14, 1914, lxiii, 20, p. 1752.

mortality of the company covering the same period and 10 per cent. higher than the general average mortality during the first five years of exposure covering the twenty years period 1885 to 1905. He further shows a mortality record of 1970 applicants rejected with an average systolic pressure of 161.44 with a mortality of 190 per cent. of the medico-actuarial table, which is more than double the average mortality of that company.

In another table are shown 1082 cases rejected in which there were reported no other impairments than high blood-pressure at the time the application was received at the home office. The expected deaths were 34.78, the actual deaths 40, or 179.53 per cent. of the table. Efforts, made to follow carefully these rejected risks in order to secure data as to the subsequent physical condition of these applicants, more than justified the opinion that the sphygmomanometer was one of the earliest, if not the very earliest, means of detecting departures from normal in this group of cases, as many impairments were later discovered or developed in a large number of cases rejected for high pressure only.

CHAPTER XXIII

THE THERAPEUTIC VALUE OF DRUGS AFFECTING BLOOD-PRESSURE AND THOSE COMMONLY EMPLOYED IN CARDIOVASCULAR AND RENAL CONDITIONS

In the revision of this work, it became apparent that it was inadvisable, if not indeed impractical, to include a discussion of the management of the many conditions in which high blood-pressure is a factor, as it would extend beyond convenient limits of size. This section is an effort to make up in a measure what to some may have appeared a regrettable omission, noting briefly the effects which may be expected to follow the employment of those drugs which are reputed to affect blood-pressure, but with no attempt to outline definite methods for the management of any specific cases.

We recognize from the therapeutic standpoint that of even greater importance than the employment of drugs is the conservation of cardiac energy, the development of circulatory competence and the promoting of elimination; and finally that the reduction of abnormally high pressures is only of secondary importance. Summing up our knowledge of the therapeutic action of the majority of drugs heretofore relied upon for the control and management of circulatory and blood-pressure changes, it may be said that our confidence in these drugs has been too great, and as a rule mistaken. Thus the experiments

made by A. Watson¹ upon the value of drugs as blood-pressure elevators were most disappointing, as he has shown that in the adult male, atropin, camphor, cotarnin, digitoxin, ergotoxin and strychnin appear to be of no value as blood-pressure elevators, while epinephrin is a dangerous drug which should always be used with great caution and never as a general blood-pressure elevator. Physostigmin is an effective blood-pressure elevator, but on account of the distressing nausea, vomiting and faintness which it produces, its use in hypotension does not seem advisable. This same general attitude toward drug therapy in blood-pressure changes is held by Janeway.² While these may be extreme opinions, they nevertheless serve to show the trend of scientific thought in the matter of the drug treatment of blood-pressure variations, as they show that we often meet failure to obtain an effect just when most urgently required. Even under the most favorable circumstances drug action is not only of short duration, but most uncertain and irregular.

In the following pages for convenience in reference the drugs considered are in alphabetic order, irrespective of their therapeutic effect or their actual value in influencing blood-pressure.

Aconite.—This drug, in its various official preparations, is usually looked upon as a vasodilator, although strictly speaking it accomplishes a fall of blood-pressure through its action upon the vagus, causing a marked slowing of the pulse which is accompanied by a reduction in blood-pressure. In overdoses a dangerous fall in pressure may

¹ *The Practitioner*, London, April, 1915, xciv, 4.

² *Jour. A. M. A.*, June 5, 1915, lxiv, 23.

be produced, accompanied by a small and rapid pulse. Its use has been recommended in interstitial nephritis, where it is said to favorably influence urea elimination.¹ It may be safely used in cases of high pressure where the heart is hypertrophied and the valves intact. It is also often employed in eclampsia with good results. In the presence of cardiac dilatation and myocardial insufficiency it is unsafe, and as compared with veratium viride, it is the more dangerous because more active.

Administration.—The tincture may be given in 5 to 10 drops every three hours; or the fluid extract 2 to 4 drops at the same intervals.

Alcohol.—Alcohol as a cardiac stimulant has little if any effect upon blood-pressure, although the capillary dilatation following its use may be accompanied by a slight fall. The moderate daily use of alcoholic beverages does not materially influence blood-pressure, although large amounts of beer, owing to the bulk of fluid ingested, may cause a temporary rise of from 5 to 15 mm.

The present feeling toward alcohol is that, as the results when used habitually may be pernicious, and as there are non-habit-forming drugs which may be used as satisfactory substitutes, alcohol should be used sparingly, if at all, in pathologic conditions, least of all in nephritis and high blood-pressure cases.

Arsenic.—This drug in doses of $\frac{1}{5}$ gr. of arsenic trioxid, has been reported by some observers, among them Balfour, to be of value in reducing high blood-pressure. To obtain an effect the drug should be administered over a long period of time.

¹ W. H. Thompson, *Am. Jour. Med. Sci.*, January, 1915, cxlix, 1.

THE NITRITE GROUP

For convenience in comparison of therapeutic effect, amyl nitrite has been taken as an example of the group of vasodilators, a number of which are discussed below. This group of drugs belongs to that large and indefinite class known as depressomotor, all of which have a distinctly sedative action upon the lower centers.

The several drugs belonging to this group, while having much in common, vary greatly in the degree and duration of their influence, while in some instances the full effect is obtained only by toxic doses. For comparison of this action, see Fig. 88. A fall in pressure following their administration is usually accompanied by increased urine excretion.

The most important vasodilators are:

Amyl nitrite.

Nitroglycerin.

Sodium nitrite (potassium).

Erythrol tetranitrate.

Mannitol hexanitrate.

Amyl Nitrite.—On account of its volatility, this drug is usually dispensed in glass pearls. These are to be crushed and the vapor immediately inhaled. The first effect of inhalation noted is hurried and panting breathing, followed by progressive muscular weakness and cutaneous flushing. Toxic doses gradually reduce reflex activity until death occurs from respiratory failure.¹

Effect of Circulation.—The pulse is increased in frequency and the arterial blood-pressure rapidly diminishes. This action is due to a dilatation of the small vessels from the

¹ H. C. Wood, "Therapeutics," J. B. Lippincott Co., Philadelphia.

direct action of the drug circulating in the blood upon the walls of the arterioles and capillaries. At the same time the drug has a minor influence on the vasomotor centers.

Administration.—This is usually by inhalation, but it may be by the mouth or hypodermically. Dose by inhalation $\frac{1}{2}$ mm.; by the mouth two to three drops on a lump of sugar to be taken instantly; hypodermically, 1 to 3 mm. The drug is comparatively free from danger; as much as two drams given within two hours have been without serious effect (Wood).

Nitroglycerin (See Fig. 88).—This drug is usually administered hypodermically, although it may be administered by the mouth in tablet form, when the same effect is obtained, although with less rapidity. There is considerable difference of opinion in regard to the proper dosage of nitroglycerin.

Von Noorden¹ gave at a single dose $5\frac{1}{2}$ gr.; Hochhaus,² 7 gr.; Himmelshach,³ $2\frac{1}{2}$ gr. On the other hand, severe collapse has resulted from comparatively small doses, as 1.12 gr. (Loeb). Initial large doses should never be given; $\frac{1}{100}$ gr. is the usual dose, although $\frac{1}{10}$ may be used with safety, though when a fall in pressures does not occur with $\frac{1}{50}$ gr., there is usually little to be expected from a larger dose.

Sodium Nitrite (See Fig. 88).—Matthiew,⁴ studying the effect of this drug on ten patients, found that in six an average fall of 30 mm. or more followed the adminis-

¹ C. von Noorden, "Discussion on Arteriosclerosis," *Cong. f. inn. Med.*, 1904, xxi, 152.

² "Discussion on Arteriosclerosis," *Cong. f. inn. Med.*, 1904, xxi, 165.

³ G. Himmelshach, *Med. News, Phila.*, 1893, lxii, 14.

⁴ *Quart. Jour. Med.*, No. 2, p. 261.

tration of 2-gr. doses. In one case a maximum drop of 75 mm. was recorded, which, however, was extremely transitory, the pressure having returned almost to the usual level in thirteen minutes, although the return to normal after employing this drug is usually slower than in nitroglycerin.

Administration.—Tablets of from $\frac{1}{2}$ to 2 gr. Solutions, when fresh, are potentially more active, but tend to decompose rapidly.

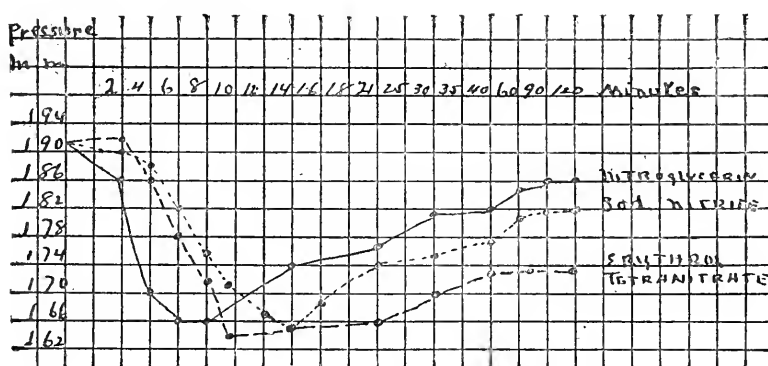


FIG. 88.—Chart showing relative effect upon systolic blood-pressure and duration of action in minutes of nitroglycerin, sodium nitrite and erythrol tetranitrate, after Miller (Jos. L. Miller, *Jour. A. M. A.*, liv, 21, 1910, p. 1666).

Erythrol Tetranitrate (Fig. 88).—The same observer following the administration of $\frac{1}{2}$ -gr. tablet obtained a fall of 30 or more millimeters in five persons, of 15 mm. in two, while one person was unaffected. In one case a maximum fall of 110 was accompanied by collapse. Headache is a common accompaniment and does not necessarily follow the degree of blood-pressure reduction.

Mannitol Hexanitrate.—The use of this drug is usually followed by a more prolonged reduction in pressure than

is produced by any of the preceding. It is usually given in doses of 1 to 2 gr.

The following table has been constructed from the clinical statistical reports of Wallace and Ringer,¹ Matthiew,² J. L. Miller,³ and Lauder Brunton.⁴

Drug	Effectual dose	Begin effect	Max. effect in	Mm. reduct.	Duration	Dose, interval
Amyl nitrite.....	1-3 mm. inhalation	1 min.	2 min.	20-40	7 min.	P. R. N.
Nitroglycerin.....	1-2 mm.	2 min.	2-10 min.	20-40	30-40 min.	1-2 hr.
Sodium and potassium nitrite..	½-2 gr.	6 min.	8-15 min.	5-30	1-½ hr.	T. i. d.
Erythrol tetranitrate.....	0.5-1.5	4 min.	15 min.	15-50	4-6 hr.	4-6 hr.
Mannitol hexanitrate.....	1 gr.	6 hr.	4-6 hr.

Before employing any drug in this group, it should be carefully ascertained that the drug, particularly sodium nitrite, is strictly fresh, as failure to obtain the desired effect may be due entirely to the use of an inactive preparation. Tablet preparations are known to vary greatly in strength and should be of standard make. This defect can, according to some observers, be avoided by the employment of fresh chocolate tablet preparations. Sodium nitrite in solution rapidly loses its activity and should not be kept for more than one week. All these drugs may be employed hypodermically when desired, but for continued use should, if possible, be given by the mouth.

According to Wallace and Ringer, it may be stated that, as a general rule, the higher the original pressure, the greater

¹ *Jour. A. M. A.*, No. 20, p. 1629.

² *Loc. cit.*

³ *Jour. A. M. A.*, May 21, 1910, liv, 21, p. 1666.

⁴ *Loc. cit.*

the fall produced. They were able in their experiments to obtain a reduction in pressure in every case, and the effect of an equal dose upon the pressure in arteriosclerosis was the same as the effect of an equal dose upon a normal individual. My own experience does not substantiate this.

Daniel Hoyt¹ arrives at the same conclusion, but advocates the use of larger doses than those generally employed, attributing failure to obtain satisfactory results to insufficient dosage or the employment of inactive preparations. This difficulty is largely removed when the clinician employs the sphygmomanometer to check his results.

Rudolph notes that the effect of the vasodilators may vary from day to day, and in this connection Miller² brought out a very interesting as well as a most important point in the clinical action of these drugs, namely, that wide variation in their effect may occur not only from day to day, but that different drugs of the same group may affect the same individual differently. He reports the following specific instances:

Case 1.—Sodium nitrite had no effect whatever, nitroglycerin caused a reduction of 50 mm., erythrol tetranitrate resulted in a rapid fall of 110 mm., the patient going into collapse.

Case 2.—Nitroglycerin and erythrol tetranitrate had very little effect upon the pressure while a reduction of 65 mm. followed the usual dose of sodium nitrite.

Case 3.—Nitroglycerin caused a fall of 30 mm., sodium nitrite a fall of 20 mm., and erythrol tetranitrate a fall of 15 mm.

¹ *Internat. Clinics*, Vol. i, 1912.

² *Loc. cit.*

Atropin.—Atropin has little if any effect on blood-pressure in normal individuals, its chief circulatory effect being to depress the vagus terminations and to accelerate the pulse. This acceleration is often accompanied by a correction of functional arhythmias.

In cases of pulmonary edema and vascular collapse this drug is of great value in controlling leaky skin and wet lungs. Here the result is as rapid as it is satisfactory (see Fig. 69).

Administration.—Usually by hypodermic, in doses of from $\frac{1}{200}$ to $\frac{1}{50}$ gr. repeated p.r.n. In the form of belladonna it is most valuable in doses of from 3 to 5 mm. in supporting the circulation, often turning the tide in the patient's favor.

Caffein.—The usual opinion seems to be that caffein causes a marked rise in blood-pressure with an increase in pulse-rate and that the rise in blood-pressure is due in part to the increased heart action, and in part to the contraction of the blood-vessels caused by a stimulation of the vasomotor centers in the medulla. The observations of H. C. Wood, Jr.¹ in a careful study of four subjects, where every effort was made to eliminate psychic and other extraneous influences, show that in the entire four there was never a marked rise in blood-pressure, the average rise for his whole series being but 2.2. mm. This was accompanied by a reduction in pulse rate, occasionally quite marked. In Wood's experiments the doses of caffein range from $1\frac{1}{2}$ to 6 gr., and were administered under such conditions that the subjects were unaware when they were receiving the drug. The protocol of Wood's experiments follows:

¹ *Therap. Gaz.*, Jan. 15, 1912.

Wood's Table Showing the Details of Two Studies on the Human
Blood-pressure
Subject I

Time	B.P.	P.P.	P.R.		Time	B.P.	P.P.	P.R.	
Begin.	84	27	53	Starch 1½ gr. at 0.25.	Begin.	88	28	57	Caffein 6 gr. at 0.20.
0.15	83	22	54		0.15	84	34	60	
0.40	87	21	50		0.45	84	30	55	
1.00	90	29	53		1.00	93	26	54	
1.10	84	23	..		1.15	93	26	54	
1.20	91	22	56		1.30	95	22	54	
					1.45	97	25	52	
					2.10	96	19	54	
					2.20	95	22	58	
					2.40	97	22	54	
					2.50	96	23	58	
					3.05	96	22	53	

Time is expressed in hours and minutes. B.P. indicates mean blood-pressure, obtained by adding together the diastolic and systolic pressures and dividing by 2; P.P. or pulse-pressure is the difference between the systolic and diastolic pressures; P.R. is the pulse-rate per minute.

Wood's Table Showing the Averages of all the Observations on the
Effects of Caffein on Circulation of Human Beings

	Normal		One-half hour after caffeine		One hour after caffeine		Two hours after caffeine	
	B.P.	P.R.	B.P.	P.R.	B.P.	P.R.	B.P.	P.R.
Subject 1.....	85	62	87	57	92	56	94	56
Subject 2.....	94	86	95	79	93	81	91	84
Subject 3.....	111	53	111	52	112	52	112	51
Subject 4.....	98	81	98	75	97	73	100	71
Average.....	97.0	70.5	97.8	65.6	99.0	65.4	99.2	65.4

From the work of J. D. Pilcher¹ it appears that caffeine in excessive doses is accompanied by a lowering of systolic pressure, accompanied by an increase in heart rate, while in toxic doses death may result from acute cardiac dilatation. The stimulating action of caffeine is more prompt but less persistent than digitalis. Coffee by mouth or by rectum,

¹ *Jour. Pharmacol. and Exp. Therap.*, July, 1912, iii, 6.

and also tea to a less extent, have the same action due to their containing this drug.

Calomel.—Lauder Brunton advises the employment of calomel in $\frac{1}{2}$ -gr. doses three or four times a day to relieve hypertension. In kidney involvement the giving of mercury in any form is usually inadvisable.

Camphor.—This drug finds great favor as a rapid and powerful cardiac stimulant, as it may be relied upon even in the last stages of cardiac failure. Experimental evidence tends to show that it has very little influence upon blood-pressure, and it is rarely employed for this effect.

Chloral may greatly relieve the symptoms of high blood-pressure, even without materially altering the level. Indeed it does not as a rule have much effect on high blood-pressure.

Chloroform.—This drug always produces a fall in blood-pressure, which progressively increases with the duration of its administration, when used in concentrated form, as little as 3 c.c. has been known to cause a dangerous fall. Its employment is dangerous in all degenerative conditions with hypotonus, and while advocated by some clinicians as an emergency remedy to reduce high pressure, it should not be used if anything else is at hand which will accomplish the same result.

Cocain.—According to Cook and Briggs, low tension after hemorrhage is favorably affected by this drug; $\frac{1}{4}$ to $\frac{1}{2}$ gr. hypodermically is usually followed by an almost immediate rise in pressure of from 10 to 20 mm., which is maintained from one to three hours.

Digitalis and Its Preparations.—Digitalis has not yet been accurately placed, at least in regard to its effect upon

the blood-pressure. Owing to its cumulative action, the marked degree of local irritation usually following the use of preparations of the crude drug, and the slowness of its action, it is of less value than it otherwise might be in acute conditions and in emergency. Further, the older preparations of this drug are so variable in their activity that they cannot be depended upon. Thus I have seen no effect follow the administration of 20-minim doses of a poor preparation and good results often accrue from 5 minims of an active tincture. This criticism applies less strongly to such modern preparations as digitoxin, digalin and digipuratum, than to the older galenicals.

Effect on Blood-pressure.—The characteristic results which have heretofore been attributed to digitalis are largely based upon experimental work and may be summarized as follows: (a) A reduction of the pulse rate, ascribed to stimulation of the inhibitory mechanism; (b) increase in the force of cardiac contraction and (c) narrowing of the lumen of the blood-vessels by the combined action of this drug directly upon the unstriated muscle and upon the vasomotor centers in the medulla. It is upon this data that the common belief that digitalis raises the systolic pressure has been founded. More recent observations by careful investigators have failed to demonstrate that digitalis can be included in the class of drugs, to be depended upon for elevation in blood-pressure. Thus Cushny¹ states that the systolic pressure of cardiac patients is rarely raised by digitalis, while on the other hand it may fall as improvement sets in. E. W. Price,² studied thirty-

¹ Arthur R. Cushny, *Am. Jour. Med. Sci.*, cxli, p. 469, 1911.

² *Brit. Med. Jour.*, Sept. 13, 1913, ii.

seven cases of which twenty-six were cardiovascular and in which the blood-pressure was either normal or sub-normal. In all except five there was no demonstrable change in the systolic pressure during the administration of the drug. Of the five cases three showed a fall equal to about 20 mm. Hg., toward the end of the administration. In one there was a doubtful fall and the fifth a rise equal to about 20 mm. This was in a case of double aortic disease with a mitral systolic murmur, thickened and tortuous vessels, cyanosis, orthopnea, slight jaundice, much edema, and slight albuminuria, and with a pressure varying between 170 and 180 systolic.

Lawrence¹ cites twenty-six cases, in only five of which was there demonstrated a rise in systolic pressure, the greatest being 30 mm., occurring in a case of acute infection involving the heart and kidneys (which cannot be considered as properly belonging to a cardiovascular series), four showed no change and seventeen showed a fall in systolic pressure, either during or immediately after the cessation of treatment, nearly all showed a marked diuresis.

Effect on Diastolic Pressure.—In Lawrence's series only one showed a relative increase in diastolic pressure, four exhibited no change whatever, while twenty showed a fall in diastolic pressure with an increase in pulse pressure, and in 50 per cent. of the cases in this group this change was accompanied by diuresis.

Administration.—In fresh infusion 5 ii to 5 iv every fourteen hours; tincture 5 to 15 m. digalin, 3 to 9 m. in twenty-four hours, digipuratum 0.1 c.c. in twenty-four hours.

¹ C. H. Lawrence, *Boston Med. & Surg. Jour.*, Jan. 1, 1914, clxx.

A. E. Taussig¹ lays down the following rules for the administration of this drug. In mitral stenosis digitalis should not be given until compensation is entirely lost and then only in moderate doses. In aortic stenosis with myocarditis the drug may be given irrespective of the character of the pulse.

There is no reason why digitalis preparations should not be safely administered to patients suffering from arteriosclerosis, angina pectoris and high pressure of nephritic origin. In the presence of decompensation, it has been my experience that the administration of digitalis preparations, in cardiovascular cases in which the myocardium is chiefly at fault, is often accompanied by danger, and that its effect should be carefully watched, and the drug immediately stopped upon the development of increasing arrhythmia or mental disturbance.

Contra-indications.—Partial heart block, recent embolism, cerebral hemorrhage, aortic aneurism, uremia, hyperthyroidism, the cardiac neuroses and in aortic insufficiency with partial heart block.

Epinephrin (*Adrenalin*).—Despite the conflicting reports upon the efficiency of adrenalin as a supporter of failing blood-pressure, a critical study shows that this drug is probably our chief support in emergency. The degree to which elevation in pressure occurs will depend largely upon the method of its administration (whether continuous or intermittent) upon the nature of the condition present and upon the ability of the arterial system to respond to its action. According to Manswetowa² as small an amount as 1 c.c. will cause a rapid and sharp rise in systolic pressure,

¹ *Interstate Med. Jour.*, xix, p. 771.

² S. M. Manswetowa, *Russky Vrach*, June 27, 1914, xiii, 24.

while the diastolic pressure falls. Thus bearing out the conclusions formerly reached by Hill.¹

Spinal Injections.—In the quantity usually employed to induce spinal anesthesia adrenalin, has no effect upon arterial pressure.

Administration.—Adrenalin may be administered by hypodermic in doses of 3 to 10 minims, and by hypodermoclysis and intravenous injections in varying dosage, depending upon the rate of flow through the needle and the extent of effect desired. The action of adrenalin when given by mouth is extremely unreliable, and it is doubtful whether absorption from the stomach takes place with sufficient rapidity to allow much of the drug to be absorbed before its activity is reduced or destroyed by the fluids in the digestive tract.

MacKenzie recommends the hypodermic method for emergency use, but he believes frequent repetition is necessary if any sustained action is desired, as the action is largely local, and because the product is rapidly destroyed after entering the blood-stream. The researches of W. Straub² confirm the assumption that adrenalin has no cumulative action. He states that it is probable that this substance is destroyed with great rapidity, as it vanishes from the blood completely, just as rapidly as its action subsides. Its action is exclusively local, that is, it acts on the vessels only by direct contact. This appears to confirm the opinion that the continuous infusion of a weak solution of adrenalin is the only rational method of employing the drug, when continued effect is desired. Straub found it

¹ E. C. Hill, *Denver Med. Times*, April, 1912, xxxi, 10.

² *Münch. med. Wochen*, Vol. lvii, No. 26.

possible to send the solution continuously into a vein and thus keep blood-pressure up permanently, as long as the drug was continued, the effect being dependent on the concentration of the solution, and not on the absolute amount of adrenalin infused.

In the low blood-pressure of shock, Pearce and Eisenbrey¹ recommend the slow intravenous administration of adrenalin salt solution (1-40,000) combined with a pure cardiac stimulant such as digitoxin. They obtained relatively rapid and permanent improvement. In this same connection A. Rendel Short² found that the addition of adrenalin to normal salt solution in strength up to 1-20,000 would restrain the caliber of the vessels even when the vasomotor center was powerless and that apparently hopeless cases recovered under this treatment.

In contrast to this testimony Brooks and Kaplan³ have reported two cases where adrenalin was used as the therapeutic agent for a prolonged time. They found that during continued administration adrenalin gradually lost its power, and they therefore do not accept the common belief that adrenalin will, over a prolonged period, maintain a constant elevation of pressure.

Ergot.—Ergot, due to its vasoconstricting effect, may be relied upon to cause a slight rise in blood-pressure. This effect contra-indicates its use in hemorrhage, particularly pulmonary.

Ethyl Chlorid.—On account of the uncertainty of the action of this drug and its marked effect in reducing systolic pressure, it should under no circumstance be employed

¹ *Arch. Int. Med.*, August, 1910.

² *Loc. cit.*

³ *Arch. Int. Med.*, Oct. 15, 1909.

therapeutically for its effect on blood-pressure, and it is especially dangerous when used as an anesthetic in the presence of myocardial insufficiency.

Guipsine (Active Principle of Mistletoe).—According to Williamson,¹ this drug is held in high esteem abroad, particularly in France, as a blood-pressure reducer, as it can be relied upon to sustain its action for a period of days. It is administered in pill form, each pill being standardized to contain the equivalent of 5 cgm. of the active principle of mistletoe and to be given in doses of from 30 to 125 cgm. in twenty-four hours; this approximates 30-mm. doses of the fluid extract of mistletoe. Toxic symptoms are extremely rare even with maximum dosage.

Hormonal.—H. Mohr² states that this substance is capable of greatly reducing blood-pressure even when employed in amounts far below the dose generally administered clinically, and the severe symptoms of collapse have been reported after the intravenous administration of 14 c.c., while 20 have caused death.

Iodin.—Iodin and the iodids are supposed to beneficially influence degenerative changes in the vessel walls and have long been advocated for the treatment of high blood-pressure, apart from those cases resulting from syphilitic infection, where of course it is indicated. The profession is, however, by no means united as to the efficiency of these preparations, which at present do not find general favor in the treatment of arterial tension. Many believe that any effect following the employment of this drug is due to the employment of coincident measures,

¹ O. K. Williamson, *The Practitioner*, London, May, 1911.

² *Wien. klin. Wochen*, May 16, 1912, xxv, 20.

much as improved hygiene, the elimination, rest, etc. One drawback to the continued use of this drug and its salts is the irritation which its use causes in the digestive tract.

Iodin is usually administered in the form of potassium

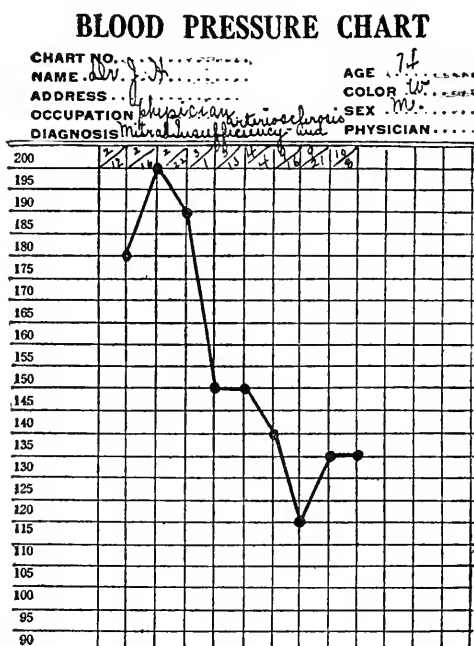


FIG. 89.—Chart shows dangerous effect of continued overuse of sodium iodid. Iodid was begun in doses of 5 gr. three times a day, and was continued until April 1. Pulse became irregular and patient was very dizzy. Strychnin, $\frac{1}{30}$ gr., was begun on April 14; patient then left city for summer. Did not return until September 16, during which time contrary to orders, he persistently took between 25 and 30 gr. sodium iodid, and returned in very bad condition. The rising pressure of September 21 is from the combined use of strychnin and digitalis.

or sodium iodid, and as there is no difference in their effect upon the circulation and, as a rule, sodium iodid is better tolerated, the sodium preparation should be employed. No advantage has been found in the use of larger doses than

2 to 5 gr. daily, given in milk or diluted with water. Some observers, however, recommend the use of an ascending dosage, beginning at 6 gr. and gradually increasing to 21 gr. a day. When used in this way an intermission of one week should occur in every four weeks of its administration. Excellent results have been reported in some cases of hypertension, but there was no proof that they were not of syphilitic origin. The accompanying chart shows a remarkable and at the same time dangerous effect from the overuse of iodid (Fig. 89).

The disagreeable effect of iodid can often be reduced by the addition of 5 gr. of sodium bicarbonate to each dose.

Morphin.—Morphin has long been recognized as a valuable emergency remedy in heart disease and there are probably no contra-indications to its use. In doses of $\frac{1}{8}$ to $\frac{1}{4}$ gr., hypodermically, it may be relied upon in emergency to lower systolic pressure and improve cardiac rhythm, but it is not a drug for continued use. The value of morphin in heart disease is probably due to its effect in breaking up the vicious circle between the heart and the nervous system.

Pilocarpin.—Robinson¹ states that both the pulse rate and the blood-pressure are reduced in the lower animals by this drug, and that while similar results obtain in man, they are not as uniform. He states, however, that he has employed pilocarpin for several years in the treatment of practically all cases of high blood-pressure with good results in most instances, and reports reduction in systolic pressure amounting to 30 or 40 mm. after four to six weeks' treatment.

¹ Wm. Duffield Robinson, *N. Y. Med. Jour.*, Nov. 7, 1914.

Dosage.—One-thirtieth of a grain in a glassful of water after meals. Even this very small dose may be further reduced and still produce good results. He cautions against over-dosage and recommends a close watch upon cardiac action, blood-pressure, and the production of sensible sweat.

Pituitary Extract.—A Rendel Short¹ considers pituitary extract more valuable than adrenalin in combating marked reduction in blood-pressure, since its action is more prolonged than that of adrenalin. It is not cumulative, and may be employed hypodermically over a long period of time. The commercial preparation of pituitrin, $\frac{1}{2}$ to 1 c.c. of the preparation, is the agent of choice; the studies of Lewis, Miller and Mathews² report variable results from preparations made from the several divisions of this gland. Remon and DeLille,³ prefer the extract and recommend $\frac{1}{5}$ -gm. doses. Arbuck and Rongy⁴ caution against the use of pituitrin in any case of high pressure in which an increase in pressure may be harmful. They found that an injection of 1 c.c. in a series of cases studied by them causes within two or three minutes after injection a rise averaging from 25 to 35 mm., which persisted from thirty to forty minutes, followed in a number of cases by a sudden fall to below the original.

Purging.—Neilson and Hyland⁵ have studied the effect of magnesium sulphate, sodium sulphate, sodium and potassium tartrate and compound jalap powder, in the

¹ *Loc. cit.*

² D. Lewis, J. L. Miller and S. A. Mathews, *Arch. Int. Med.*, June, 1911.

³ Quoted by Sajous, *loc. cit.*

⁴ S. S. Arbuck and J. A. Rongy, *N. Y. Med. Jour.*, May 2, 1914, p. 878.

⁵ C. H. Neilson and R. F. Hyland, *Jour. A. M. A.*, Feb. 8, 1913, lx, 6, p. 436.

usual therapeutic doses, from which they noted the following effects:

The systolic pressure was tested in 126 patients, of whom 109 showed a lowering of the systolic pressure varying from 5 to 35 per cent. Twelve showed practically the same blood-pressure throughout the experiment. Five showed an increase during the experiment. Twenty-four hours after the cathartic was given, forty-eight had a systolic pressure from 5 to 18 per cent. lower than before the cathartic was given. Twenty-six had practically the same as at the beginning. Only those who remained in bed were tested twenty-four hours afterward. The action of the different cathartics was practically the same, except that the compound jalap produced a more constant and greater lowering than the others. They also showed in most instances quite a decided lowering at the end of twenty-four hours. It was found in this set of experiments that those individuals with a systolic pressure of 140 and above, the highest tested being 190, gave an average lowering of 23 mm. Hg. Fifty-seven individuals with a pressure of from 110 to 140 gave an average lowering of 13 mm. Hg., which thirty-four individuals with pressure 110 or lower gave an average lowering of 7 mm. Hg.

A study of systolic, diastolic and pulse pressures, and the rate of heart beat was made on sixty-eight individuals.

The percentage lowering of the systolic pressure in this number taken as a whole, was 17 per cent.

The diastolic pressure was lowered 8 per cent.

The pulse pressure was decreased 24 per cent.

The number of heart beats as a whole were decreased

14 per cent. Fifty-six patients showed a decrease, nine showed an increase and five showed no change.

If we examine these results we find that an average lowering for all cases is 18 mm. Hg. The average lowering for those who had initial pressures ranging between 140 and 190 was 23 mm. Hg.

Those individuals who had a low initial pressure lowered only 7 mm. Hg.

For instance, one fell from 180 to 100; another from 170 to 120; another from 190 to 115; another from 120 to 80; another from 108 to 78, etc. It is in these extreme results that we most frequently find the development of an arrhythmia or the increase of an arrhythmia already present.

The result of this set of experiments, supported by the clinical fact that patients with diseased hearts may become worse on brisk catharsis, warrants the assumption that all cases, in which severe purging is used for depletion of the blood, ought to be controlled by watching carefully the blood-pressure, heart rate and regularity, and the general condition of the patient.

Salicylates.—All salicylates in large doses reduce blood-pressure but are rarely employed for this effect, although Hamburger¹ recommends their use for this purpose, basing his opinion upon the results of experiments with intravenous and hypodermatic injections of sodium salicylate in dogs.

Acetylsalicylic Acid.—The author has for some time employed this preparation for the control of morning headaches so frequent in high-pressure cases, usually administering 5 gr. upon rising, and has been able to demon-

¹ *Interstate Med. Jour.*, 1911, p. 667.

strate to his satisfaction that not only are these headaches controlled with this minimum dose, but a continued reduction in systolic pressure is obtained, and it is not usually necessary to repeat or increase this dose.

Strophanthus.—See *Digitalis*.

Strychnin.—There has been considerable controversy concerning the effect of strychnin on blood-pressure, although MacKenzie in "Diseases of the Heart" has held that this drug has no appreciable effect on blood-pressure. Recently Wallace and Pemment¹ after a careful investigation of the effect of this drug upon blood-pressure, conclude that in approximately normal pressures the drug fails to produce a rise, except when employed in doses too large to be considered therapeutic. In artificially produced low pressures they obtained the following results: After chloral, strychnin hastens the return to normal level; after nitrites there is no appreciable improvement; after hemorrhage there is no effect; after diphtheria toxin there is no effect; after chloroform no effect; after shock by traumatism to intestines there is no effect. They conclude from this series of experiments that the only type of low pressure favorably affected by strychnin is that following depression of the vasomotor center, such as is brought about by chloral.

Thiosinamin.—Lydston² recommends the employment of this preparation for the reduction of high pressure in which relief is obtained by relieving arterial contraction, but states that it is not of value in effecting improvement

¹ G. B. Wallace and H. C. Pemment, *Jour. A. M. A.*, July 20, 1912, lix, 3, p. 218.

² G. F. Lydston, *Therap. Gaz.*, July, 1912, p. 466.

through alterations in structural change. The dose is $\frac{1}{5}$ to $\frac{1}{2}$ gr. three times a day.

Thyroid.—The relation of the ductless glands to the maintenance of normal blood-pressure and to disturbance of the same, through abnormalities in internal secretion, is too well known to require comment, except to say that it has been repeatedly pointed out that a deficiency in thyroid secretion may contribute to an elevated blood-pressure, while an increase in adrenal secretion is believed to have the same effect, and that signs pointing to such abnormality should always be sought for.

Administration.—In cases of high pressure, without excessive cardiac or renal involvement, this drug may prove of great service in reducing and maintaining a more normal pressure level. It has also been found of value in the high pressures of eclampsia. Small doses of the dried gland, 1 to 3 gr. per day, are usually as efficient as larger doses, and large doses should never be used, nor the drug be continued for a long period of time, unless the blood-pressure and pulse rate are kept under close observation.

Tobacco.—There has always been considerable doubt as to whether the effects ascribed to tobacco were due to nicotin or to other substances. The majority of evidence indicates that nicotin is by far the most important, if not the only factor concerned in elevating blood-pressure, although the by-products of the dry distillation of tobacco, such as furfural and CO must be reckoned with. Nicotin is, next to adrenalin, the most powerful vasoconstrictor known. Cook and Briggs¹ have shown that a temporary

¹ *Loc. cit.*

rise in systolic pressure accompanies smoking, and yet we frequently have the apparent paradox that excessive smokers often have a low systolic pressure. In the habitual smoker (Fig. 90) it is found that there is at first

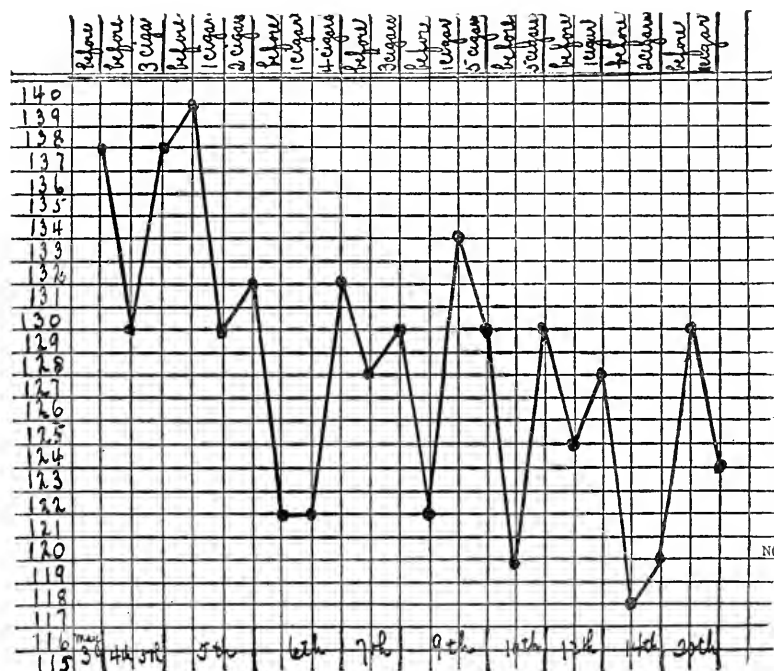


FIG. 90.—Showing the effect of moderate smoking on one accustomed to the use of tobacco, after a brief period of abstinence. Tracing shows very well the sedative effect of a moderate amount of tobacco, and the pressure-raising influence of several cigars smoked in rapid succession. Also the general downward tendency of the curve would suggest that the individual became gradually reaccustomed to the use of the drug.

an increase and later a diminution in systolic pressure, the latter being accompanied by that sense of comfort and relief which is sought by the habitual user. The reduction in pressure following the use of tobacco is from 5 to 15 mm. From the experimental standpoint, it does not appear

that smoking is an etiologic factor in the production of arteriosclerosis, at least, in so far as such a theory presumes injury to the vessels.¹ The use of tobacco in those unaccustomed to it is often accompanied by a sharp systolic rise.

Vasotonin (*Urethan and Yohimbin*).—Muller and Fellner,² report both animal experiments and clinical observations of the effect of this drug on blood-pressure. They conclude that its effect is purely that of peripheral dilatation and state that, while the drug is generally used abroad, the results so far in this country have not been uniform.³ One of the most valuable effects of this drug is said to be its prolonged action; thus the subcutaneous injection of 1 c.c. is usually sufficient to reduce blood-pressure from 20 to 40 mm., for a period of from four to six hours, while if given for several successive days it will maintain a lowered pressure for six or seven days thereafter.

Veratrum Viride.—This drug is classified with the heart depressants. Its chief physiologic action is upon the circulation, and in practice it is used chiefly to decrease the force of the heart. It is "a prompt, thoroughly efficient, and at the same time very safe remedy" (Wood).

In chronic cardiac diseases it is indicated in precisely those cases in which digitalis is contra-indicated. The contra-indications to the use of this drug are cardiac weakness and general adynamia. When used in excess it may cause alarming symptoms which simulate shock, but even in very large doses it is seldom fatal (Wood). In this respect it is far less dangerous than aconite. Its physio-

¹ Bruce, Miller and Hooker, *Amer. Jour. Physiol.*, April, 1909.

² *Therap. Monat.*, 1910, xxiv, p. 285.

³ H. D. Arnold, *Boston Med. and Surg. Jour.*, 18.

logic effect is shown in a slow pulse rate, a diminished force of the heart's action, and vasodilatation.

Administration.—Fluidextract, one to three drops, tincture three to six drops. It should be given at intervals of two or three hours, when continued effect is desired, and its activity may be hastened by gradually increasing the dose until the physiologic limit is reached. In some cases annoying vomiting may occur.

CHAPTER XXIV

PHYSICAL MEASURES EMPLOYED IN THE MANAGEMENT OF CONDITIONS PRESENTING ABNORMAL BLOOD-PRESSURE

Causes of Failure.—A large percentage of the unsuccessful results in the treatment of cardiovascular-renal diseases can be traced to one or more of the following causes:

1. The diagnosis has not been made sufficiently early.
2. The case may have been carelessly or unskilfully studied.
3. The predisposing causes have not been found.
4. As a result, the condition is but imperfectly understood.
5. The therapy is irrational because it is based upon an incomplete knowledge of the case in question, plus a deficient knowledge of therapeutic methods by drugs or other measures.
6. Too great dependence has been placed upon drugs alone, especially the vasodilators, to the neglect of the newer so-called physiologic methods.

It may be said in general that while drugs are at times invaluable in the treatment of pathologic circulatory conditions, especially in emergency, their value is usually much overestimated. The secret of successful treatment usually lies in a careful study, an early and complete diagnosis, rigid supervision and regulation of the individual habits, rather than attempts to lower blood-pressure and relieve symptoms by the employment of drugs. A properly conducted study will sometimes yield gratifying

results even in advanced cases, and at times in those cases commonly regarded as hopeless.

The most satisfactory results naturally follow an exhaustive examination immediately following the appearance of the first suggestive sign or symptom of impairment of the circulatory apparatus. This should be followed by a careful estimate of the functional power left in the impaired organs and the immediate adoption of a life and habits suited to the limitations determined. Thus we attempt to produce an adjustment of the individual's manner of life which will give an equivalent to relative good health. By "correct diagnosis" the broadest meaning of this phrase is intended and not the mere statement that the patient has "cardiovascular-renal disease."

To arrive at a correct diagnosis, one must take a full history, including a complete analysis of social history and personal habits, carefully considering both business and social activities, making a comprehensive physical examination, including blood and urine examinations, and taking the blood-pressure, not omitting the functional tests. In fact the success of treatment depends primarily upon the completeness in which each problem is studied and upon the intelligence with which the remedies are employed and only in a minor degree upon the particular remedial measures applied.

Direct therapeutic measures aimed at distinct pathologic conditions will not be considered, as they are beyond the object and scope of this book. This chapter will consist more of a résumé of existing literature and will be more in the nature of a reference chapter to be consulted when knowledge of the relative value of certain measures

and information concerning the effect of any particular drug is desired.

PHYSICAL MEASURES

Under the head of physical measures valuable in controlling and in reducing high pressure, we find:

Rest.

Exercise.

Massage.

Diet.

Hydrotherapy.

Electrotherapy.

Venesection.

Rest and Posture.—Preëminently rest is the first essential in the treatment of all cardiovascular and renal conditions. It is safe and beneficial to begin every course of treatment by rest. The term rest as here used may be purely relative or may mean absolute recumbency. The degree of rest enforced will depend on the physician's judgment as based upon experience and the extent of his knowledge of the case and its requirements; no set rule can be adhered to blindly.

In the cases suddenly developing signs of incompetency, with dyspnea, a large heart, venous congestion, etc., the decision is obvious; absolute rest and mental relaxation are imperative—nothing else will do. First and foremost, all unnecessary strain must be removed from the overburdened and dilated heart. This alone may suffice to break the vicious circle, allow the heart muscle to regain its lost tone and so pave the way for a period of at least relative health.

Rest in bed will alone often be sufficient to reduce a dangerously high blood-pressure. I have repeatedly seen a pressure of over 200 mm. fall to and maintain a new level of from 15 to 25 mm. lower. Occasionally even a greater reduction than this will be effected by this measure.

Effects of Sleep and Rest on Blood-pressure.—Brooks and Carroll¹ studied this question in sixty-eight patients showing average systolic pressure, in thirty with low pressures and in twenty-nine with abnormally high pressures. The results are, in a general way, illustrated in the cases with average pressure, in which readings taken between one and two hours after the beginning of sleep showed an average drop of 24 mm. Hg. For three hours after awakening in the morning there was still an average depression of 12 mm. and from this time onward the pressure gradually rose until usual highest level was reached in the afternoon. The greatest nocturnal fall in pressure took place in those individuals having the highest initial systolic reading. Disturbance of patients during the first sleep was found to delay, but not necessarily prevent, the maximal fall in pressure; frequent interruption did, however, prevent it. Special tests were made to determine whether the sleep drop could be artificially increased in order to secure a lower general pressure curve in cases of hypertension; potassium bromid in doses as high as 120 gr., and chloral hydrate up to 50 gr. each night were not found to increase the degree or persistence of the fall. Physical rest in general did not appear to alter materially either supernormal or normal blood-pressure, but Brooks and Carroll

¹ *Archives of Int. Med.*, August, 1912.

were led to believe that in mental or psychic rest profound changes in pressure occur, and that this factor largely determines the undoubted benefit derived from rest in cases of high pressure.

Exercise.—In certain cases, particularly those of the active business and professional men, it is not more rest but more exercise that is needed. These are the cases in which, if seen sufficiently early, much may be accomplished toward permanently arresting the trouble, provided of course that the patient is ready and willing to assume a new rule of life. They probably belong among those classed as true hypertonus, with tonic contraction of the circular fibers of the arteries (see page 244), with but little or no permanent pathologic change, the kidneys showing only signs of irritation. Under these conditions complete relief often follows a carefully regulated diet, combined with an increased amount of daily exercise, which should not be begun suddenly, nor be very strenuous; walking first, to be followed later by light gymnastics or golf. Such measures should always be carefully followed by sphygmomanometer tests.

In institutions and hospitals devoted to the treatment of chronic cardiovascular and renal diseases the exercise methods of Schott and Ortel, under competent supervision and proper guidance, accomplish much good by educating the heart muscle to withstand added strain and by improving cardiomuscular tonus. It is not advisable that the general practitioner, seeing but few cases, should attempt these special exercise treatments, as they need specialized attention. However, much can be accomplished by systematized walking.

Massage.—General massage is usually well borne and is of value in the treatment of cases showing failing compensation or defective heart tone. Massage acts by emptying the venous side of the circulation and so relieves the left side of the heart; it also dilates the superficial capillaries, thereby aiding in the distribution of the blood. Massage of the chest may influence favorably the tone of the heart itself, but deep pressure upon the abdomen should be avoided in order to escape a rise in blood-pressure and all movements should be graded to the strength of the individual.

Both Eichberg¹ and A. Strausser² advocate the employment of massage in the treatment of cardiovascular diseases, and Eichberg has shown that massage movements, even if prolonged, do not effect a rise in blood-pressure.

Dietetics.—Much has been said and many dietetic outlines have been advocated in the treatment of circulatory disturbances. Their chief object is to diminish nitrogenous intake, to reduce putrefactive changes in the intestines which produce auto-intoxication, and to relieve the strain on a dilated and defective heart muscle by reducing dangerously high pressure through limiting the fluid intake which eventually modifies the total amount of fluid in the body.

Foods.—A safe general rule to follow is, that while nitrogenous food is not prohibited, the amount should be greatly reduced, and a vegetable, farinaceous and milk diet substituted.

An absolute milk diet cannot be continued over a long

¹ *Jour. A. M. A.*, Sept. 19, 1908.

² *Wien. med. Wochen.*, Apr. 8-15, 1909.

period because it is impossible to give sufficient nourishment without seriously overstepping a safe maximum of fluid ingestion, excessive fluid sometimes being a factor in the production of high blood-pressure.

A short period of absolute milk (2 quarts) is useful for the relief of certain symptoms, and may, if guarded, be employed with benefit. It should be given at two- or three-hour periods and never in large quantities at one time. The addition of some flavoring or prepared junket renders the milk diet less irksome to the patient.

In the treatment of cardiovascular cases the best results generally follow light meals taken at frequent intervals (three to three and one-half hours). This prevents possible harm from the throwing of heavy strain on the heart and blood-vessels through the digestive apparatus, which might easily disturb a poorly balanced circulatory equilibrium.

Alcohol, tea and coffee are usually prohibited, at least for a time. An exception to this would perhaps be a heavy drinker, whose habitual potations should not be suddenly and entirely interdicted. As a substitute for coffee, postum may be employed; and recently a patented process has been used in Germany by which the coffee bean is freed of 90 per cent. of its caffeine. The taste of the coffee is not materially changed, but the effect upon the heart and blood-vessels is decidedly lessened. Elsner¹ and others report the use of this preparation during a period of several years to their satisfaction.

Tobacco.—Tobacco in the form of pipe, cigarettes and cigars has the power of raising blood-pressure, with the seeming paradox that the habitual smoker has ordinarily

¹ *Boston Med. and Surg. Jour.*, No. 7, 1910.

a low pressure. Arterial disease tends to augment the effect of smoking on arterial pressure. It is often a point of delicate decision to determine the amount of harm resulting from the use of tobacco, and the proper restriction in the use of the drug necessary in each particular case. When in doubt the best rule is to regulate the patient's habits, with due regard to special idiosyncrasies. In cases with a history of anginoid attacks, tobacco in all forms should be prohibited entirely.¹

The habit of chewing tobacco is much more harmful than smoking because of the greater amount of active principle, nicotin, which enters the system. It should not be tolerated.

In restricting diet, no definite rule can be laid down which can be followed safely in every case. Each case has its own peculiarities and the physician should endeavor to determine the restrictions to be made, and what things may be allowed with safety in a given case. One should be careful in any dietetic scheme to avoid a caloric reduction below the needs of the individual, otherwise much harm may be done, for it is impossible to build up a strong heart upon insufficient nourishment.

L. F. Bishop² makes the following suggestions which may serve as a valuable guide in the preparation of a diet list in hypertension and chronic heart disease.

First, he suggests that every student of the subject should address the Superintendent of Documents, Government Printing Office, Washington, D. C., enclosing ten cents and asking for *Bulletin* No. 28 on the "Chemical Composition of American Food Materials."

¹ A. Strausser, *Wien. klin. Wochen.*, Apr. 15, 1909.

² *N. Y. Med. Jour.*, Mar. 4, 1911.

Second, the principle to be remembered is that an adult required from 14 to 20 calories per pound, body weight, proportional to his physical activities, the weight to be estimated by the normal weight for the height of the individual. For example, a person 5 ft. 7 in. tall ought to weigh 150 lb.; at light work he will require an average number of heat units per pound 17, $150 \times 17 = 2550$ calories. If a healthy man averages more than this, he will accumulate fat; if he has less he will become run down, and a weak heart cannot be built up on insufficient nutrition.

Bishop submits the following dietary covering a period of five days, which allows a fair caloric intake:

DIET		
	Calories	Protein
<i>Luncheon:</i>		
1 cup of bouillon.....	40	10
2 slices of mushroom on toast.....	50	2
1 tablespoon of potatoes.....	100	2
1 plate of endive and lettuce salad.....	125 (oil)	
1 saucer of rhubarb.		
1 piece of gingerbread.....	230	4
<i>Dinner:</i>		
1 plate of vegetable soup.....	50	3
3 tablespoons of stewed tomatoes.		
1 large tablespoon of potatoes.....	110	2
2 large tablespoons of beans.....	60	4
2 tablespoons of Indian pudding.....	175	8
Lactose with each meal.....	300	
<hr/>		
Average breakfast.....	315	8
<hr/>		
Total for day.....	1565	43
<hr/>		
JANUARY 21.		
<i>Breakfast:</i>		
1 orange.....	40	
1 small bowl of wheat berries.....	160	4
2 slices of toast.....	115	4
1 cup of weak coffee.		

Luncheon:

4 large fried scallops.....	60	8
2 tablespoons of creamed potatoes.....	220	4
1 plate of cabbage and lettuce salad.....	125 (oil)	
2 tablespoons of preserved peaches.....	40	
1 cup of weak tea.		

Dinner:

1 plate of vegetable soup.....	50	3
3 small slices of bread.....	230	8
2 tablespoons of potatoes.....	220	4
2 tablespoons of spinach.		
1 plate of scallop and lettuce salad.....	140	4
1 small piece of pumpkin pie.....	250	4
1 small piece of cheese.....	120	8
Lactose with each meal.....	300	
<hr/>		<hr/>
Total for day.....	2070	51

JANUARY 22.

<i>Breakfast</i> , practically as before.....	315	8
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Dinner:

1 plate of vermicelli soup.....	120	4
4 small potatoes.....	200	4
2 tablespoons of gravy.		
3 stewed onions.....	100	4
Ice cream.....	320	8
Cake.....	230	4
3 small slices of bread.....	230	8

Tea:

1 cup of bouillon.....	40	10
1 tablespoon of fried potatoes.....	110	2
3 slices of bread.....	238	8
1 plate of lettuce and celery salad.....	125 (oil)	
1 cup of weak tea.		
Ice cream.....	320	8
Sponge cake.....	230	4
Lactose with each meal.....	300	
<hr/>		<hr/>
Total for day.....	2870	72

JANUARY 23.

<i>Breakfast</i> , as before.....	315	8
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Luncheon:

3 tablespoons of macaroni.....	100	3
2 tablespoons of spinach.....		
3 small slices of bread.....	230	8
1 plate of lettuce and endive salad.....	125 (oil)	
1 piece of pumpkin pie.....	250	4
2 pieces of cheese.....	120	8
1 cup of weak tea.....		

Dinner:

Large plate of farina soup.....	50	2
3 tablespoons of macaroni.....	100	3
2 tablespoons of potatoes.....	220	4
3 pieces of preserved peaches.....	40	
3 slices of bread.....	230	8
Lactose with each meal.....	300	

Total for day.....	2080	48
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JANUARY 24.

<i>Breakfast</i> , as before.....	315	8
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Luncheon:

1 plate of lettuce and endive salad.....	125 (oil)	
2 tablespoons of potatoes.....	220	4
2 tablespoons of fried hominy.....	120	4
3 pieces of preserved peaches.....	40	
1 cup of weak tea.....		

Dinner:

Large plate of vegetable soup.....	50	3
2 tablespoons of boiled potatoes.....	180	4
2 tablespoons of stewed peas.....	100	7
2 tablespoons of rice pudding.....	175	4
Lactose with each meal.....	300	

Total for day.....	1625	34
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N. B.—One glass Sauterne with each luncheon.

Average for five days: Protein, 49; calories, 2,040.

Roughly speaking the average helping of meat contains 25 gm. of proteid, an egg contains 8 gm., as does also a glass of milk. An ordinary helping of rice, potatoes, bread or hominy contains about 4 gm., thick cream, butter and oil contain practically no proteid, but are very rich in heat units. Green vegetables do not count one way or the other. In cardiovascular disease, milk sugar is a valuable addition to diet, for many reasons. A sufficiency of calories can be roughly judged by watching the weight; if the weight is maintained the caloric supply is sufficient.

Water.—Water properly employed may be of great value in the treatment of cardiovascular and renal diseases, but like any other good thing, it can be overworked. Cases are on record where apparently the only causative factor in the production of chronic interstitial nephritis was continued excessive water drinking. Usually it is advisable to limit the amount of water, especially in extreme high-tension cases or where there is a tendency to edema, for this will spare both the heart and blood-vessels, but the amount should not be reduced below 1500 c.c. per day. Even when there is edema, the reduction of water should not be continued for more than three consecutive days below this figure (A. Strausser).

HYDROTHERAPY

As before stated, the functional capacity of the heart depends entirely upon the condition of the heart muscle and not in the slightest degree upon disease of the valves. So in the therapeutics of chronic heart disease, estimation of the integrity of the muscle is of first importance as our

chief aim is to restore that muscle as near as possible to its functional integrity.

Hydrotherapy in the treatment of high blood-pressure, particularly accompanied by arteriosclerosis, accomplishes its result chiefly through regulation of the circulation. Properly used, such methods may under certain conditions check the progress of disease by breaking the vicious circle in which the patient is involved. The primary effect of plain water, either hot or cold, applied to the surface of the body, has been found by most observers to cause an initial rise in blood-pressure.

This elevation usually amounts only to a few millimeters and is followed speedily by a reaction, accompanied by lower pressure, due to a relaxation of hypertonus and diminished peripheral resistance, resulting in an increased flow of blood through the capillaries.

Cold Baths.—A cold shower constricts the skin and lowers the axillary and mouth temperatures to normal or sub-normal. The pulse is reduced in frequency but increased in volume and the blood-pressure is elevated. The breathing volume and alveolar tension is increased.

The application of cold to the precordium is immediately followed by a rise in the systolic pressure.¹

The careless application of cold to high-pressure cases may be dangerous. The effect should first be ascertained by rubbing cold water over portions of the body. Cold applications can only be used with safety in early arteriosclerosis and cold douches should be used with extreme caution, as they do not as a rule give as good results as rubbing or ordinary bathing. Cold sea-water baths should

¹ Hirschfeld and Lewin, *Zeitsch. f. Physiol. u. Diet. Therap.*, No. 1, 1914.

not be indulged in by arteriosclerotics nor by those having myocardial degeneration.

The Scottish douche (alternate application of hot and cold water) frequently gives good results in hypertension, provided the contrast between the temperatures employed is properly graduated.

Hot Baths.—A full hot bath (105 to 110°F.) raises the mouth, axillary and rectal temperatures and is accompanied by an increased pulse frequency (up to 160) and a lowered systolic pressure. The rise in body temperature is accompanied by greatly increased respiratory frequency and breathing volume, and is accompanied by a notable fall in carbon-dioxid tension, and a corresponding rise in the oxygen tension of the alveolar air.¹

Because of the constant and fairly uniform reduction in systolic blood-pressure accompanying the hot bath, it is believed to be a reliable adjunct in the treatment of many of the chronic diseases in which there is high blood-pressure. It also acts as a powerful diaphoretic and this action seems to be of some value in these cases.² According to Hirschfeld and Lewin³ the application of local heat causes a rise in systolic pressure.

The temperature of hot baths in cases of arteriosclerosis should not exceed 37 or 38°C. Extreme changes in temperature of baths is also contra-indicated in arteriosclerosis, because of the danger in any sudden change in pressure, particularly any sudden increase arising from capillary contraction which causes increased peripheral resistance.

¹ Leonard Hill and Morton Flack, *Jour. Physiol.*, xxviii, p. 441, 1909.

² C. L. Palmer, *Penna. Med. Jour.*, March, 1915, xviii, 6.

³ *Loc. cit.*

Hot-air baths and electric-light baths are probably as good as the direct application of hot water, and should be employed whenever practical.

In the high blood-pressure accompanying acute nephritis, with the usual subjective symptoms, I have seen great benefit follow a properly given electric-light bath, the temperature being allowed to rise in the cabinet to 125°F. where it is maintained for from fifteen to twenty minutes. Under these circumstances an immediate fall in pressure occurs, often amounting to from 15 to 30 mm., occasionally more, and this fall is usually lasting in character, often persisting for twenty-four hours. The effect upon the patient is most satisfactory, the subjective signs immediately subsiding. Elimination is increased while the pathologic elements in the urine are diminished.

The proper administration of an electric-light bath depends upon the intelligent use of the sphygmomanometer. By this instrument, and by its aid alone can the immediate effects of the bath be measured, so that the duration and the period of administration may be definitely calculated. Hydrotherapeutic measures sometimes accomplish good results when drug medication absolutely fails. This was well shown in one case, where nitroglycerin was given to the point of intolerance, without effect upon blood-pressure, while the electric-light bath speedily reduced the pressure and easily maintained a reduction of 45 mm.

Miller recently has reported a practical series of clinical studies on the effect of the sweating process in high-pressure cases. In his series all patients reported sweated profusely for at least thirty minutes. The methods of producing the sweat varied. The blood-pressure was taken just before

the sweat ceased. Three out of five cases showed a reduction in pressure ranging from 13 to 20 mm. In one case it did not return to previous level until a lapse of four hours. Patients always felt better after the sweating. Dyspnea (uremic) is generally relieved even when the pressure is not reduced.

A number of patients were given one or more daily sweats for two or three weeks, the pressure recorded daily; results varied, in some there was no change, some showed a gradual fall. One case which had been over 210 for several years, came down to 180.

In chronic cases the sweating process is not lasting in its effect, as the pressure soon returns to original level when sweats are discontinued. Poststernal oppression is relieved more often by sweats than by other measures.

Effect of Sweating on Blood-pressure (Miller)

Case	Blood-pressure before sweating	Blood-pressure after sweating	
1	160	140	Four hours before it reached previous level.
2	190	190	
3	170	170	
4	190	175	Two hours before it reached previous level.
5	185	172	

The sudden application of cold or chilling after a sweat is dangerous. In one case Miller has reported a rise of 60 mm., followed by transitory numbness. Over-reduction of pressure may be followed by untoward results, although this does not always follow.

The Nauheim Treatment.—There is no method of determining in advance the particular effect of any special hydrotherapeutic measure. This applies to low-pressure

cases as well as to those with a high initial pressure, so that the relief of subjective symptoms in cases of heart disease is not necessarily dependent upon the influence of the baths upon blood-pressure. The basis of the Nauheim treatment in circulatory disorders is rest, hydrotherapeutic measures and exercise. Its chief value in the treatment of circulatory disorders comes from its effect on the heart muscle. Acting upon the heart, it increases tonus and reduces dilatation. Acting upon the circulation, it dilates the arterioles and capillaries, thereby relieving a high peripheral resistance and obtaining a more uniform distribution of blood. These baths do not always produce a reduction in blood-pressure, and they may be followed by disastrous results. In this connection it is important to sound a warning note. Neither the oxygen nor the CO₂ bath should be used without a working knowledge both of what is desired and what such treatment may be expected to accomplish.

Application.—The chief hydrotherapeutic method employed at Nauheim is the complete immersion of the patient in a bath of natural brine, which is charged with free CO₂ gas. The most important constituents of this bath are sodium chlorid and calcium chlorid. The temperature of the bath is varied according to experience. The patient remains immersed for a period of from four to fifteen minutes, is then carefully dried, without chilling, and required to rest in bed for an hour. The baths are given on alternate days; the course usually occupies six weeks. Baths of similar character are given under medical supervision at Glen Springs, N. Y., where the methods are much the same as those at Nauheim and the benefits derived probably as great.

According to Dr. John M. Swan,¹ formerly of Glen Springs, the effects to be expected from the proper use of carbonated-brine baths are as follows:

1. Diminution of the size of the heart.
2. Slowing of the pulse.
3. Reddening of the skin.
4. Slowing of the respiration.
5. Reduction in the size of the liver, if that organ has been the seat of passive congestion.
6. Improvement in the muscular quality of the heart sounds.
7. The disappearance of hemic murmurs, or those due to dilatation of an orifice.
8. Increase in the intensity of those murmurs which are dependent on valvular defect or deformity.

The chief indication for the use of the carbonated-brine bath in the treatment of chronic heart disease is in cases of myocardial weakness, with low pressure. In such cases we expect to get a retarding of the pulse, an improved heart-muscle sound and a rise in blood-pressure.

In cases of senile heart, with high blood-pressure and evidence of general arteriosclerosis, carbonated-brine baths, if given at all, should be stopped upon the development of an increase in blood-pressure, whether shown by subjective symptoms, or by the sphygmomanometer. In cases where the beneficial effect of the bath is in doubt, danger may be avoided if the temperature of the bath is kept above 98°F., or else the strong brine should be omitted or diluted, and the CO₂ gas left out. According to Swan, CO₂ baths are contra-indicated in cases of advanced arteriosclerosis, chronic nephritis, aneurism of the large arterial

¹ *Interstate Med. Jour.*, June, 1911, xviii, 6.

trunks, and in the terminal stages of broken compensation with edema.

After the diseased myocardium has had an opportunity to recuperate, and to regain some of its lost tone by rest and the bath treatment, it is often advisable to provide exercise under proper supervision in order to help the heart perform its normal functions in as nearly a normal fashion as possible. This is in the nature of a special training of the muscles to be developed. Two systems have been devised which apply graduated work to the heart: first, Schott method or resistance movement; and, second, Ortel method of graduated hill climbing. These consist of a number of exercises of increasing severity, arranged so that the increased work imposed on the heart is very slight, but is increased in proportion as the heart muscle learns to bear the strain. For more complete descriptions of these methods and their application, the reader is referred to works devoted to hydrotherapy and the treatment of heart diseases.

Oxygen Bath.—A mode of treatment that has recently been advocated and favorably reported upon by a number of observers is the oxygen bath. According to reports the effect of the oxygen bath is very different from that of the CO_2 . In the CO_2 bath the skin becomes reddened from dilatation of the superficial vessels, while in the oxygen bath the cutaneous vessels are constricted and the skin becomes pale. The oxygen bath at 95°F . reduces both pulse rate and blood-pressure, while the effect of the CO_2 bath upon blood-pressure is variable. In arteriosclerosis these baths are said to be beneficial, resulting among other things in a moderate reduction of blood-pressure. According to the conclusions of A. Wolfe¹ the respective effects of

¹ *Zeitsch.f. Physiol. u. Viet. Therap.*, Vol. xiv, 1910.

the oxygen and the CO₂ bath upon the human body are as follows:

1. The temperature of the water in both instances has a material bearing upon its influence on blood-pressure.

2. At 93 or 94°F. neither bath has much influence on blood-pressure if this be not pathologically changed. The CO₂ bath at 94° tends primarily to increase a pathologic blood-pressure, whether this was at first a hypo- or a hypertension.

3. The normal pulse is but little altered by either bath, while the CO₂ reduces it more often in less degree than the oxygen bath, when the pulse is originally abnormal.

In employing the oxygen bath, the patient should not enter it immediately after active exercise or mental excitement, and unnecessary movement should be avoided while in the bath. He should be carefully dried and then should immediately lie down for an hour. The duration of the bath, depending upon the effect desired, should be from ten to twenty-five minutes, and should be given on alternate days. The bath is contra-indicated in low blood-pressure accompanying the last stages of arteriosclerosis. Also for those with mitral defects or marked anemia.

The ingredients for the oxygen bath (sodium perborate and magnesium borate) can be obtained in the open market under the name of "perogen" bath.

ELECTROTHERAPY

Much has been said, and, if possible, more has been written, upon the subject of electrical treatment for the reduction of arterial hypertension. A careful review of literature up to the time of writing shows that there is

considerable divergence of opinion on the value of such measures. First, in any case, we must determine the cause of high pressure and the desirability of reducing it.

Cornwall¹ warns against too promiscuous employment of high-frequency treatments in high-pressure cases, particularly in arteriosclerosis, in nephritis, and in the toxic types. He bases his position in the matter upon the ground that any agent may do harm which lowers blood-pressure without removing the factor or factors that cause it.

Here, as in the study of other remedial agents, a systematic employment of the blood-pressure test is essential to the proper interpretation of the results, as it is only by this means that the psychic element can be eliminated, which, some authorities aver, is the only benefit derived from the use of electrical currents in the treatment of hypertension.

William Benham Snow² is conservative in his statements regarding the value of such measures, and confines himself chiefly to the consideration of the control of early cases of hypertension by autocondensation and other electrical measures.

He divides all cases presenting the symptoms of hypertension into the following seven clinical groups:

1. The aged and feeble, partly compensated arteriosclerotics with low-pressure readings. (These are not benefited by electrical treatment—author.)

2. General arteriosclerosis, so widespread that autocondensation fails to affect the reading, sequelæ cannot be avoided and electrical treatment is useless.

¹ Edward E. Cornwall, *Jour. Med. Res.*, Nov. 16, 1912.

² *Jour. Adv. Therap.*, June, 1909.

3. Arteriosclerosis of advanced age, fifty to sixty years, pressure above 200 mm.; autocondensation and hygienic measures may cause a reduction to 165 or 160 mm., where it may be maintained by diet and occasional electrical treatment. There is a corresponding improvement in general health. Electric treatment is valuable in this class if it can be continued indefinitely at stated intervals in order to maintain the reduction.

4. Arteriosclerosis in adults of thirty-five to fifty-five, pressure 150 to 170 mm., with or without beginning chronic nephritis. Here fifteen-minute treatments, 400 milliamperes by autocondensation, produce marked fall; with frequent treatments and correction in diet the tension often returns to normal, the physical condition appears normal and urine clears up. (These cases are probably those of true hypertension of Brunton, where there is no permanent arterial change or chronic intestinal nephritis—author.)

5. Same as class four, except an earlier stage of hypertension—(author).

6. Young adults, chiefly athletes, who have developed a work hypertrophy and consequent moderate degree of hypertension. (Snow fails to state effect of treatment—author.)

7. Compensatory hypertension occurring in parenchymatous nephritis, cirrhosis of liver, fever, after excessive exercise, etc. (Condition about the same as 4, no uniformity in results of treatment—author.)

Snow states that D'Arsonval high-frequency and static-wave currents act locally upon the neuromuscular mechanism. The method of D'Arsonval may be either autocon-

densation or autoconduction, by both of which methods the patient is placed in a field of hypotensive stresses where the high frequency to a greater or lesser extent surges through the tissues of the body, and is remarkably active in lowering arterial tension. "This effect is probably induced by a complex action of the current." Acting conjointly:

1. Upon metabolism, promoting tissue combustion and elimination, as demonstrated by an increase in solids in the urine, and

2. Upon the vasodilator centers which control peripheral resistance by which hypertension is relaxed, as demonstrated by the sphygmomanometer.

A twelve-minute administration of 400 milliamperes is, as a rule, followed by a reduction of from 10 to 15 mm.; occasionally a fall amounting to 50 occurs.

"Autocondensation is indicated in all cases in which hypertension is not compensatory and is contra-indicated in all compensatory cases" (Snow).

Dosage 300 to 400 milliamperes from twelve to fifteen minutes' duration, repeated daily or on alternate days.

Van Allen¹ claims that high-frequency currents reduce the blood-pressure by removing the exciting causes, that is, by preventing auto-intoxication

Hirsh² is a warm advocate of the electric treatment of high blood-pressure and believes that in certain cases the blood-pressure even when very high is apparently the sole factor and that these cases are relieved of their symptoms only by a reduction of their high pressure.

¹ *Albany Med. Ann.*, June, 1911.

² A. B. Hirsh, *Penna. Med. Jour.*, December, 1914, xviii, 3, p. 189.

He cites a number of cases where the D'Arsonval treatment was the only measure employed, and relates the following characteristic results: The reduction of the systolic pressure from 220 to 135 mm. with the relief of symptoms said to be of more than thirty years' duration. A case of chronic parenchymatous nephritis with systolic pressure of 240 (an almost unprecedented pressure in this type of nephritis) was reduced to 160. A case of early arteriosclerosis with a pressure of 180 reduced to 130. A case of a woman sixty-one, diagnosis not made, the systolic pressure of 190 reduced to 120, with "complete cure." A case of arteriosclerosis with cardiac hypertrophy and a pressure of 185, reduced in two weeks to 155 and kept there by an occasional treatment. A case of very marked sclerotic and tortuous vessels, reduced from 210 to 150. In the face of adverse criticism, this is an almost startling array of evidence, upon which the author makes no comment, it still being his belief that, owing to the present status of electrical treatment, when a definite diagnosis based upon a well-established pathology can be made, it would be safer to employ the usually accepted methods and reserve the electrical measures for those cases which are apparently unaffected by the usual methods.

It must be remembered that all efforts at reduction of high blood-pressure should be based upon a carefully made diagnosis, and that the indications for interfering with the circulation must be clear; otherwise failure may be expected, for in some cases disaster will follow ill-advised efforts to modify blood-pressure. A safe rule is to watch the patient, study the effect of pressure changes upon him and cease all measures that fail to produce benefit,

either in the evident physical condition of the patient or in the subjective signs.

VENESECTION

Miller,¹ after carefully studying the effect of venesection on both normal and pathologic cases, arrived at a conclusion similar to that stated by Mackenzie some years before. Miller found that the rapid withdrawal of 300 c.c. or more from a normal individual is followed by a transitory fall in blood-pressure, but that all persons do not react in the same way, the effect depending partly on the degree of rapidity with which the blood is withdrawn, as 500 c.c. withdrawn slowly may have no effect on blood-pressure.

Effect of Bleeding on Blood-Pressure (Miller)

Case	Blood-pressure before bleeding	Amount of blood withdrawn, c.c.	Blood-pressure after bleeding	
1	200	500	200	
2	190	500	185	
3	160	600	150	Two hours later 160
4	185	500	170	Two hours later 180
5	220	450	210	

Butterman² bled ten students, withdrawing from 200 to 480 c.c. and nine showed reduction varying from 5 to 30 mm. Patients with hypertension do not necessarily all react in the same way.

The accompanying table taken from Miller's article mentioned above shows what may be expected in efforts to reduce hypertension by this means.

¹ *Jour. A. M. A.*, Vol. liv, 21.

² *Arch. f. klin. Med.*, 1902, lxxiv, 1.

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